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OF THE
# AMERICAN VETERINARY MEDICAL ASSOCIATION





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# "CORN STATES"

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## 1914-1939

***celebrates its Silver Anniversary or twenty-five years devoted to the conservation of live stock and farming.***

To accomplish that exclusive purpose as well as is humanly possible, the directive factors were:

***(1) Belief in the veterinary profession***
***(2) Reliance in college-trained personnel***
***(3) Up-to-date production technique***
***(4) Modern laboratory appointments***
***(5) Classical distribution and delivery***
***(6) Uniform treatment of all patrons***

With these intentions uppermost in mind, "Corn States" enters nineteen thirty-nine with faith in the future of the profession it has the privilege to serve and wishes its members the

*Compliments of the Holiday Season*

The CORN STATES SERUM COMPANY
OMAHA

# JOURNAL
*of the*
# American Veterinary Medical Association

Established 1877

Editorial Office, 221 N. La Salle St., Chicago, Ill.

H. PRESTON HOSKINS, Editor — L. A. MERILLAT, Executive Secretary

COMMITTEE ON JOURNAL

H. W. JAKEMAN, Chairman

H. D. BERGMAN — O. V. BRAMLEY




Vol. XCIV, N. S. Vol. 47 — JANUARY, 1939 — No. 1

## VITAMIN STUDIES PRODUCE IMPORTANT RESULTS

Probably never before in the history of vitamin studies have vitamin-A, vitamin-$B_1$ and vitamin-D deficiencies and the black-tongue condition been produced in a group of dogs and exhibited in one place at the same time. This was done and the dogs were shown as a part of the Small-Animal Scientific Exhibit at the Diamond Jubilee meeting of the American Veterinary Medical Association, held in New York City, July 5-9, 1938.

The dogs were carefully selected and clinical examinations made before placing the animals on the deficient diets. During the production of these deficiencies, records were kept of the body-weight changes. Vitamin supplements were administered to correct the diets for every known factor except the particular deficiency to be produced. The growth, general condition and clinical appearance of these dogs were carefully noted, and daily records were kept on the condition of the appetite and bowels. Temperatures were recorded at intervals. In addition, blood-count and blood-chemistry findings, as considered important from the standpoint of differential diagnosis, were obtained.

Interest in this exhibit was such that the Executive Board of The American Veterinary Medical Association decided to continue the studies and determine the therapeutic value of the respective vitamins when administered to the dogs showing symptoms of the deficiencies. Quantitative supplements of the several vitamins were administered and observations made of the effect upon the dogs. Still and motion pictures were made of the subjects at various times during the experiment.

An interesting feature of this demonstration was the time required to produce simultaneously all of the deficiencies so that each animal would be showing characteristic symptoms between the 5th and 9th of July. To accomplish this it was necessary in certain instances to maintain the animals on a minimum daily requirement of the deficient vitamin.

Since this work was done coöperatively by a veterinarian and a worker in nutritional research, it will be possible to present the results from both the clinical and scientific standpoints. Reports on these experiments are to be published in forthcoming issues of the JOURNAL.

## SULFANILAMIDE

The dynamics of sulfanilamide and related drugs have been studied extensively. The germicidal properties of these compounds have been measured quite accurately; the harmful action upon certain cellular elements and organs has been calculated carefully; and the list of bacterial infections, for which these drugs are now regarded as the classical treatment, has been enlarged gradually and catalogued. Summed up, these studies lead to the same end. The blood and kidneys can be damaged, depending upon the capacity of the body to handle the drug.

To prescribe the drug intelligently, one must determine the concentration in the blood and the rate of clearance by way of the urine as the dosing proceeds. In short, sulfanilamide, the main example of its class, can produce hemolytic anemia and grave intracellular edema of the glomeruli. That the anemia caused by the drug is hemolytic was proved by Harvey and Janeway.[1] The character of the renal damage is common knowledge. The cause is a preëxisting nephrosis and the result a more or less serious oliguria, sometimes amounting to complete suppression of the renal secretion.

Unfortunately, these two sequels of sulfanilamide therapy are not easily avoided. Both may occur from moderate dosage. Tolerance is not predictable from the casual physical examination. The erythrocyte count and hemoglobin may drop to a low level from small doses, or they may remain unaltered from large ones. In one observation made by Wood,[2] at Johns Hopkins Hospital, the incidence of anemia was 4 per cent and, in another, it was 8.3 per cent. There was no apparent reason for the impoverished blood.

An important point brought out by these observations, however, was the fact that the anemia was more pronounced in febrile than in afebrile cases. The red-cell destruction followed doses of 0.2 grams per kilogram of body weight in some instances and did not occur at all in others receiving twice that amount. Hence, the cause of sulfanilamide anemia is some predisposing factor, not the dose of the drug. A rise of the body temperature of more than three degrees (F.) seems to be a warning to check the red-cell count after the first few doses or before irreparable injury has been inflicted. Mere withdrawal may not overcome the erythropenia the drug has produced. Blood transfusion may be required. In 21 cases described by Wood,[2] 13 required from two to four transfusions to restore the normal red-cell level. The lysis is therefore grave. In regard to renal edema, the seriousness corresponds to that of the preëxisting nephrosis.

Another fact coming to light is the overrating of sulfanilamide as a urinary antiseptic. The work of Gaudin, Gide and Thompson[3] on the use of the drug to clean up the urinary tract following transurethral prostatectomy seems to prove that there is no sound basis for sulfanilamide therapy in urinary infections.

These facts are pointed out for the purpose of keeping a useful drug in its right place in animal medicine, where uncontrolled clinical reports are apt to be deceptive. More than two years ago, the Council on Pharmacy and Chemistry of the American Medical Association warned physicians against the indiscriminate use of sulfanilamide. The warning still stands and it applies to all branches of medicine.

[1]Harvey, A. M., and Janeway, C. A.: Development of acute hemolytic anemia during administration of sulfanilamide (para-aminobenzene sulfonamide). Jour. Amer. Med. Asso., cix (1937), pp. 12-16.

[2]Wood, W. B., Jr.: Anemia during sulfanilamide therapy. Jour. Amer. Med. Asso., cix (1938), pp. 1916-1919.

[3]Gaudin, H. J., Gide, H. A., and Thompson, G. J.: The use of sulfanilamide after transurethral prostatectomy. Jour. Amer. Med. Asso., cx (1938), pp. 1887-1890.

## IT'S YOUR HOUSE OF REPRESENTATIVES

No matter in what branch of the service you labor, the House of Representatives of the American Veterinary Medical Association is your court of last resort in organized veterinary medicine. And, when each affiliated association chooses wisely its delegate to the House and sends him to the annual meeting instructed to exercise his constitutional authority, this body will become the dictator of the veterinary profession on this continent.

Quoting from section 12 of article V of the Constitution:

> The House of Representatives shall exercise all of the duties and assume all of the powers heretofore delegated to the active members of the Association by the Constitution and By-laws, except the election of the President, Vice-Presidents, Treasurer and members of the Executive Board.

This provision for electing officers the members retain as their vested right.

Speaking relatively, this legislative body is new—too new to be well understood, too new to perform well the functions for which it was created, too new even to comprehend fully its purpose, too new to know its power over the officers and members. Such a body, like all living things, must be born and pass through youth and adolescence to maturity before it can strike the stride expected of it.

The founding of such a legislative body in the national association was conceived soon after the turn of the century, and its make-up was discussed by the Executive Committee (now the Executive Board) from year to year. The pattern of the American Medical Association was the inspiration, but a survey of the situation throughout the United States and Canada showed that the membership of the state, provincial and territorial organizations did not justify the attempt or father the thought. Later, or about 1915, when the state associations began to enlarge their memberships, the idea took form. The late lamented C. A. Cary pioneered the plan and never ceased to nurture it until the present House of Representatives became a reality. The details of its organization were the work of the late T. E. Munce, of Pennsylvania.

Elsewhere in this issue, we list the personnel of the House of Representatives for 1938, that is, the delegates who served at the New York meeting—the Diamond Jubilee of the Association.

The House is composed of one representative and one alternate for each affiliated organization, chosen by election or appointment for a term of two years. While the representative must be a member of the A. V. M. A. in good standing, all members of his association may participate in his selection.

As the time for the annual meetings of many of the state associations is approaching, and as the importance of making the House the power it is intended to be is unquestionable, presiding officers, secretaries and program committees of the state organizations are urged to give the selection of their representatives a prominent place on the "order of business" and, thereby, seize the opportunity of making the House the ruler in the empire of organized veterinary medicine, and the delegate chosen their faithful servant.

In short, the sooner the various associations choose wisely their delegates to the national association, the sooner all branches of the veterinary service will be cemented into a solid unit capable of moving forward toward the profession's objective.

## THE SYNDROME OF ENCEPHALOMYELITIS

By means of intracranial injections of suspensions of brain tissue containing the specific encephalomyelitis virus, Mitchell, Walker and McKercher* have clearly outlined the symptomatic genesis of the disease. They established six days as the period of incubation, and a sudden rise of the temperature up to 103° F. as the first symptom. During the following 18 to 24 hours, a hyperthermia of 104 to 106° F. developed, only to decline gradually, however, during the succeeding twelve hours. These preliminary phenomena they named the "early febrile stage."

The first physical evidence of the disease, superseding the practically inapparent onset, the authors named the "hypersensitive stage," because of the pronounced hypersensitivity of the special senses of feeling and hearing. This stage, which lasted about twelve hours, was followed by the "early lethargic stage," manifested by opisthotonos, muscular tremors, engorged mucosae, and fetid breath. The fourth, or "late lethargic stage," comprises the terminal phenomena leading to convalescence or death, in short, to the well-known symptoms of the developed case.

*Mitchell, C. A., Walker, R. V. L., and McKercher, D. G.: The clinical symptoms of encephalomyelitis in artificially infected horses. Can. Jour. Vet. Med., ii (1938), pp. 271-275.

## ON REORGANIZATION

At the mid-year meeting of the Executive Board, held in Chicago, November 28, 1938, several matters of vital importance to the Association were acted upon. Routine business of the Board and a discussion of plans for increasing the activities of the Association, thus making it an organization of greater value and service to a larger number of veterinarians, resulted in an exceptionally busy session.

The Special Committee of the Executive Board held three meetings in Chicago between the New York convention and the Executive Board meeting, studying problems connected with the reorganization program and endeavoring to effect an organization of the central office which would more satisfactorily meet its increased activities and responsibilities. We believe that the recommendations of the Committee, as approved by the Board, constitute a decided step forward.

To avoid conflict with the present Constitution and By-laws and pending their revision, temporary titles have been employed for the personnel of the new official set-up in the central office.

Dr. L. A. Merillat has been appointed as Executive Secretary and Dr. H. Preston Hoskins as Editor of Publications.

Dr. Hoskins, for many years, has been carrying a burden beyond that which might reasonably be expected of any individual. His knowledge of A. V. M. A. affairs is greater than that possessed by anyone and it is invaluable to the Association, especially at this time of developing a forward-looking program which will culminate in the A. V. M. A. more effectively and efficiently meeting the needs and demands of internationally organized veterinary medicine. A Department of Publications is being planned which will, in addition to one or more journals, from time to time, publish pamphlets on various subjects for the information and use of members. Dr. Hoskins will now have more time to devote to this very important development work and, with a staff of associate editors, made up of outstanding veterinarians, much will be accomplished.

Mr. Julius Shaffer, a young man of ability, enthusiasm and industry, is working in the Chicago office as assistant to Dr. Hoskins.

The Association has been fortunate in securing the services of Dr. L. A. Merillat as Executive Secretary. Dr. Merillat needs

STAFF OF THE A. V. M. A. SECRETARIAT

(Back row, left to right) Ruth Andersen, secretary to Dr. Hoskins; Cecil Harris, A. V. M. A. membership records and accounts; Adele Ray, files and lists; Wanda Landwehr, Journal accounts and mailing; Jean Weinert, secretary to Dr. Merillat. (Front row) Dr. H. Preston Hoskins, Editor of Publications; Dr. L. A. Merillat, Executive Secretary; Julius Shaffer, Assistant Editor.

no introduction to the membership. He is a past president, a former secretary and, since 1928, has been an active, conscientious member of the Executive Board. For over 50 years, he has been actively engaged in various fields of veterinary endeavor—practitioner, teacher, author, historian, commercial executive, popular speaker, active in veterinary associations and a veterinarian with the welfare of the profession always foremost. As Chief Veterinarian of the First Army, during the World War, he established an enviable record.

While much more might be said about our Executive Secretary, it is unnecessary, as Dr. Merillat's record is well known to practically all veterinarians. The fact that we have been able to secure the services of a man with such wide experience and knowledge in veterinary affairs should prove reassuring for the future of the A. V. M. A.

It is proposed to employ some recent graduate in veterinary medicine who has an outstanding scholastic record and who possesses necessary qualifications in other respects, such as public speaking, personality and evidence of executive ability. This young man will enter the A. V. M. .A. office as an understudy. He will be expected to learn the various functions of the office and the Association, so that he can fill any vacancy which might occur.

H. W. JAKEMAN,
*Chairman—Executive Board.*

## A. V. M. A. EXHIBIT FUND

Three additional contributions have been reported by Dr. J. R. Mohler to the American Veterinary Medical Association Exhibit Fund:

| | |
|---|---|
| Maine Veterinary Medical Association | $ 25.00 |
| National Association of B. A. I. Veterinarians | 100.00 |
| Veterinary Medical Association of New Jersey | 50.00 |

The amount previously reported* was $772.50. The three contributions listed above bring the total to $947.50.

*Journal of the A. V. M. A., December, 1938, p. 353.

## CONVENTION DATES

Acting on a recommendation made by the Committee on Local Arrangements for the Memphis meeting, through Dr. John H. Gillmann, Chairman, the A. V. M. A. Executive Board, at the special meeting held in Chicago, on November 28, 1938, fixed the dates for the 1939 convention as:

**August 28-29-30-31-September 1**

DR. JOHN H. GILLMANN
Chairman of Committee on Local Arrangements for Memphis meeting.

The first day, Monday, August 28, will be given over to meetings of the Executive Board and various committees and the first session of the House of Representatives. The opening session of the convention will be on Tuesday morning, August 29. The closing day, Friday, September 1, will be devoted to the clinic.

## National Safety Council Cites Veterinarian's Injury

Among the strange mishaps of 1938, as pointed out recently by the National Safety Council, the following was reported:

Dr. Verne A. Scott was operating on a calf's eye at the John Tarleton Agricultural College, Stephenville, Texas. The calf remained calm, but a 200-pound student watching the operation fainted and fell against Dr. Scott, thereby causing him to cut himself.

# APPLICATIONS FOR MEMBERSHIP

The applications given first listing this month complete the list of those filed during the calendar year 1938. As pointed out last month, we did not do so well in 1938 as compared with 1937. However, this should make it easier for 1939 to surpass 1938 in the number of new members enrolled.

If each non-member of the A. V. M. A. would appreciate the fact that he stands to gain every time a constructive move is made by the national organization, he soon would realize that he has an obligation to perform—to support properly organized veterinary medicine, not only national but state and local. At the present time, the obligation to the A. V. M. A. entails an expenditure of only five dollars per year—the annual dues.

Twice each year we publish the section from the By-laws covering the manner in which an application shall be made. Here it is:

> Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

ALLISON, LT. AARON FRANCIS
Federal Bldg., Memphis, Tenn.
D. V. M., Kansas State College, 1938. Vouchers: Col. B. A. Seeley and Lt. Col. S. G. Kielsmeier.

BIXBY, JOHN S.
531 S. Holmes St., Memphis, Tenn.
D. V. M., Washington State College, 1932. Vouchers: John H. Gillmann and C. P. Branigan.

CUNNINGHAM, CHARLES HENRY
Live Stock Sanitary Service Laboratory, University of Maryland, College Park, Md.
D. V. M., Iowa State College, 1938. Vouchers: E. J. McLaughlin and William R. Crawford.

EDMONDSON, HARRY K.
1801 Homan Ave., Fort Worth, Tex.
D. V. M., Indiana Veterinary College, 1920. Vouchers: G. R. Louden and C. J. Young.

DENNEY, PAUL C.
27 S. Barksdale, Memphis, Tenn.
D. V. M., Kansas State College, 1918. Vouchers: John H. Gillmann and C. P. Branigan.

DIGRANES, JOSEPH HAAKON
336 Federal Bldg., Oklahoma City, Okla.
D. V. M., Iowa State College, 1934. Vouchers: L. J. Allen and Wilbur McPherson.

HATCH, RAY D.
Iowa State College, Ames, Iowa.
D. V. M., Iowa State College, 1937. Vouchers: W. G. Venzke and H. L. Foust.

JOHNSON, KLEMENS FRANK
511 University Ave., Laramie, Wyo.
B. S., D. V. M., State College of Washington. 1938. Vouchers: Aubrey N. Lee and F. H. Melvin.

JONES, CARREL L.
1435 Murphy's Lane, Salt Lake City, Utah.
D. V. M., Kansas State College. 1912. Vouchers: E. D. Leiby and W. T. Huffman.

KALLENBERG, EDWIN FRANK
Union City, Tenn.
D. V. M., McKillip Veterinary College, 1917. Vouchers: John H. Gillmann and O. B. Neely.

KIME, WILLIAM
613 Dominion Public Bldg., Winnipeg, Man., Can.
V. S., Ontario Veterinary College, 1905. Vouchers: R. H. Lay and W. Moynihan.

LUCAS, J. W.
Abingdon, Ill.
D. V. M., McKillip Veterinary College, 1916. Vouchers: John D. Reardon and L. A. Merillat.

MACKENZIE, WILLIAM PARKER
Franklin Roads, Maine.
V. S., B. V. Sc., Ontario Veterinary College, 1938. Vouchers: Allan J. Neal and J. F. Witter.

NICHOLS, WILBERT C.
Box 447, Payette, Idaho.
D. V. M., State College of Washington, 1938. Vouchers: E. E. Wegner and A. K. Kuttler.

PUNDT, WERNER
1709 East Main St., Medford, Oregon.
B. S., D. V. M., State College of Washington, 1938.
Vouchers: Joseph M. Arburua and E. W. Cantrall.

RUTH, VINCENT W.
Box 1671, Charleston, W. Va.
V. S., B. V. Sc., Ontario Veterinary College, 1938. Vouchers: S. E. Hershey and H. M. Newton.

SOLDNER, PAUL ALBERT
State and North Sts., Dover, Del.
D. V. M., Ohio State University, 1938. Vouchers: B. N. Lauderdale and T. H. Applewhite.

WILSON, JOHN THOMAS
Pawnee, Okla.
D. V. M., Kansas State College, 1910. Vouchers: L. J. Allen and Wilbur McPherson.

YABSLEY, FRANCIS CHARLES
Cissna Park, Ill.
D. V. M., Chicago Veterinary College, 1917. Vouchers: W. B. Holmes and A. E. Dickerson.

## Applications Pending

SECOND LISTING

(See December, 1938, JOURNAL)

Brown, James N., Box 242, Nassau, N. P., Bahamas.

Butterworth, J. A., Box 273, Highland Park, Ill.

Carroll, Howard F., 1328 Portola Drive, San Francisco, Calif.

Chadwick, Vernon D., 507 Federal Bldg., Little Rock, Ark.

Chapin, Chalmer W., 23 S. 97th St., Belleville, Ill.

Crandall, Nelson D., University of Missouri, Columbia, Mo.

Dykstra, Lewis A., Lena, Ill.

Fitch, James A., 504 Pokegama Ave. N., Grand Rapids, Minn.

Glover, A. D., Jr., Box 138, National Stock Yards, Ill.

Harris, Frank C., 1096 Biltmore, Memphis, Tenn.

Merrick, Andrew C., 9115 Ogden Ave., Brookfield, Ill.

Nunez, Fernando Camargo, Nogal No. 226, Mexico City, Mexico.

Owens, Karl R., Mayo, Fla.

Railsback, Lee T., Harrison, Ark.

Sprinkle, W. C., 1241 N. 8th St., Terre Haute, Ind.

Traylor, David H., 420 Edgewood Ave. N. E., Atlanta, Ga.

Von Gremp, C. C., Decatur, Ga.

The amount which should accompany an application filed this month is $10.00, which covers membership fee and dues to January 1, 1940, including subscription to the JOURNAL.

## CONGRESSMAN GILLIE

Veterinarians generally will be pleased to know that a member of their profession has been elected to Congress. Dr. George W. Gillie, of Fort Wayne, Indiana, was elected to the Seventy-sixth Congress on November 8, 1938, to represent the Fourth

DR. GEORGE W. GILLIE

Congressional District of Indiana. He was the Republican candidate and was elected by a comfortable majority in a district that is normally Democratic. Dr. Gillie is a graduate of Ohio State University, class of 1907, served as meat and dairy inspector of Allen County, Indiana, until 1914, when he entered practice. He served as sheriff of Allen County for six years. In 1920, he was the Republican candidate for mayor of Fort Wayne, but was defeated.

Our best wishes, Congressman Gillie.

Great is the force of habit; it teaches us to bear labor and to scorn injury and pain. —*Cicero.*

# COMING VETERINARY MEETINGS

Small Animal Hospital Association, Los Angeles, Calif. January 3, 1939. Dr. R. W. Gerry, Secretary, 8474 Melrose Ave., Los Angeles, Calif.

Pennsylvania, Conference of Veterinarians at University of. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. January 3-4, 1939. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 3-6, 1939. Dr. Chas. J. Parshall, Secretary, 319 B St., Petaluma, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. January 4, 1939. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 4-6, 1939. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Dallas-Fort Worth Veterinary Medical Society. Adolphus Hotel, Dallas, Texas. January 5, 1939. Dr. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. January 5, 1939. Dr. E. G. Pigman, Secretary, 4206 Polk Ave., Houston, Texas.

Minnesota State Veterinary Medical Society. Hotel Lowry, Saint Paul, Minn. January 9-10, 1939. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 9-10, 1939. Dr. F. Y. S. Moore, Secretary, McAlester, Okla.

Intermountain Livestock Sanitary Association. Salt Lake City, Utah. January 9-11, 1939. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. January 10, 1939. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Rhode Island Veterinary Medical Association. Hotel Narragansett, Providence, R. I. January 10, 1939. Dr. J. S. Barber, Secretary, 310 State House, Providence, R. I.

New Jersey, Veterinary Medical Association of. Hotel Hildebrecht, Trenton, N. J. January 10-11, 1939. Dr. J. G. Hardenbergh, Secretary, c/o Walker-Gordon Laboratory Co., Plainsboro, N. J.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 10-12, 1939. Dr. Chas. C. Dobson, Secretary, New Augusta, Ind.

Maine Veterinary Medical Association. Dr. M. E. Maddocks' hospital, Augusta, Me. January 11, 1939. Dr. A. E. Coombs, Secretary, Skowhegan, Me.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. January 11, 1939. Dr. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R St., Detroit, Mich. January 11, 1939. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Willamette Valley Veterinary Medical Association. Chamber of Commerce rooms, Hillsboro, Oregon. January 11, 1939. Dr. T. Robert Phelps, Secretary, Oregon City, Ore.

Wisconsin Veterinary Medical Association. Madison, Wis. January 11-12, 1939. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 12-13, 1939. Dr. W. A. Hagan, Dean, New York State Veterinary College, Cornell University, Ithaca, N. Y.

Kansas City Veterinary Medical Association. Kansas City, Mo. January 16, 1939. Dr. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Building, Balboa Park, San Diego, Calif. January 16, 1939. Dr. Glenn A. Tucker, Secretary, Vista, Calif.

District of Columbia Veterinary Medical Association. Mayflower Hotel, Washington, D. C. January 17, 1939. Dr. E. Barnwell Smith, Secretary, 222 C St., N. W., Washington, D. C.

Iowa Veterinary Medical Association. Hotel Des Moines, Des Moines, Iowa. January 17-19, 1939. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. January 18, 1939. Dr. Chas. Eastman, Secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

Tennessee Veterinary Medical Association. Noel Hotel, Nashville, Tenn. January 23-24, 1939. Dr. A. C. Topmiller, Secretary, c/o Department of Agriculture, Nashville, Tenn.

North Carolina State College Short Course for Veterinarians. University of North Carolina, Raleigh, N. C. January 23-26, 1939. Dr. C. D. Grinnels, State College, Raleigh, N. C.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 24, 1939. Dr. R. A. Mays, Secretary, 415 State Office Building, Columbia, S. C.

Texas, State Veterinary Medical Association of. Adolphus Hotel, Dallas, Texas. January 24-25, 1939. Dr. M. B. Starnes, Corresponding Secretary, 202 City Hall Annex, Dallas, Texas.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. January 25, 1939. Dr. C. S. Rockwell, Secretary, 4927 Osage Ave., Philadelphia, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Copley Square, Boston, Mass. January 25, 1939. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Kentucky Veterinary Medical Association and Conference for Veterinarians at University of Kentucky. University of Kentucky, Lexington, Ky. January 25-26, 1939. Dr. W. W. Dimock, Head, Department of Animal Pathology, University of Kentucky, Lexington, Ky.

Mississippi State Veterinary Medical Association. Hotel Forrest, Hattiesburg, Miss. January 26-27, 1939. Dr. E. H. Durr, Secretary, Clinton Blvd., Jackson, Miss.

Ontario Veterinary Association. Royal York Hotel, Toronto, Ont. January 26-27, 1939. Dr. W. J. Rumney, Secretary, 612 King St. W., Hamilton, Ont.

Nevada State Veterinary Association. Reno, Nev. January 27, 1939. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Missouri Veterinary Medical Association and University of Missouri Veterinary Short Course. University of Missouri, Columbia, Mo. February 1-3, 1939. Dr. C. L. Campbell, Secretary, 7224 Tulane St., Saint Louis, Mo.

Hudson Valley Veterinary Medical Society. Poughkeepsie, N. Y. February 8, 1939. Dr. J. G. Wills, Secretary, Delmar, N. Y.

Alabama Veterinary Medical Association and Short Course for Graduate Veterinarians. College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. February 12-13, 1939. Dr. I. S. McAdory, Secretary, Alabama Polytechnic Institute, Auburn, Ala.

Kansas Veterinary Medical Association. Veterinary Division, Kansas State College, Manhattan, Kan. February 15-16,

1939. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Illinois State Veterinary Medical Association. Leland Hotel, Springfield, Ill. February 16-17, 1939. Dr. C. C. Hastings, Secretary, Williamsville, Ill.

Colorado State College Veterinary Short Course. Fort Collins, Colo. February 21-23, 1939. Dr. Jas. Farquharson, Colorado State College, Fort Collins, Colo.

Louisiana Veterinary Medical Association and Louisiana State University Veterinary Short Course. Dalrymple Memorial Building, Louisiana State University, Baton Rouge, La. February 22-23, 1939. Dr. C. M. Heflin, Secretary, Box 1933, Baton Rouge, La.

## State Board Examinations

Connecticut Board of Veterinary Registration and Examination. January 3, 1939. Further information may be obtained from Dr. Geo. E. Corwin, Secretary, State Capitol, Hartford, Conn.

Oklahoma Board of Veterinary Medical Examiners. January 11-12, 1939. State Capitol Building, Oklahoma City, Okla. Further information may be obtained from Dr. W. C. McConnell, Secretary, Holdenville, Okla.

## United States Civil Service Examination

The United States Civil Service Commission has announced an open competitive examination for the position of Assistant Parasitologist (nematodes) in the Bureau of Animal Industry, Department of Agriculture, at a salary of $2,600 per year.

The necessary application forms may be obtained from the Secretary, Board of U. S. Civil Service Examiners, at any first-class post office, or from the U. S. Civil Service Commission, Washington, D. C. Applications must be on file not later than January 10, 1939 (east of Rocky Mountains), or January 13, 1939, for western points.

## On the Sick List

Dr. Cassius Way, president-elect of the A. V. M. A., visited Chicago the latter part of November for the purpose of attending the meeting of the Executive Board of the A. V. M. A. on November 28, and a number of other meetings during that week. He was obliged to return to his home in White Plains, N. Y., earlier than he had planned, on account of illness. His physicians sent him to Saint Agnes' Hospital in White Plains, where he was operated upon, on December 10, for a cicatricial ulcer, located at the pyloric end of the stomach, producing a partial stenosis of the pylorus. The condition was pronounced non-malignant. Following the operation, Dr. Way developed an ether pneumonia and blood transfusions, twice daily, became necessary. By December 20, his condition had improved to the point where his physicians were hopeful for a complete recovery.

Dr. I. E. Newsom, of Fort Collins, Colo., member of the Executive Board of the A. V. M. A. for District 6, suffered an attack of angina pectoris on December 19, and was sick for about four days. The latest report from Fort Collins is to the effect that his condition is much improved, following confinement to his home and expert medical attention.

## U. S. L. S. S. A. Report

For the past ten years, the JOURNAL of the A. V. M. A. has published the proceedings of the annual meetings of the United States Live Stock Sanitary Association in the March issue following each meeting. At the 1938 meeting of the A. V. M. A., it was decided to discontinue publishing these proceedings as a regular issue of the JOURNAL. Therefore, the only form in which the proceedings of the 1938 meeting of the U. S. Live Stock Sanitary Association will be available will be the report now in the process of being published, a copy of which will be mailed to each member. If you have not paid your dues this year, send your check for two dollars to the Secretary, Dr. L. Enos Day, 3933 Drexel Blvd., Chicago, Ill.

# Streptococcic Mastitis and Public Health*

*By* PAUL B. BROOKS, *Albany, N. Y.*
*Deputy Commissioner of Health*

As science is just in its infancy and we are only beginning to get an intelligent understanding of the human body and its ailments, it is perhaps not remarkable that we do not know more than we do about the cow's udder, its diseases and their relation to public health. Three years ago, I searched the literature and corresponded in vain in an effort to find out whether there were bacteria and leukocytes in the udder before the first lactation. No one seemed to know. A little before that time, a medical graduate, taking a state licensing examination in hygiene, said that bovine mastitis is a disease of the cow's "rudder." Today, I believe, most doctors of human medicine know that the cow's milk-secreting organ is an udder and not a "rudder"; yet I doubt if one in ten has more than a vague idea of the relationship between mastitis and human illness. To a group of veterinarians, I presume that comes as a great surprise. But, after all, we are beginning to "gain on it."

At the outset, I would like to emphasize the fact that streptococcic infection is not the only infection of the bovine udder with which we are concerned from a public health standpoint. However, it is the most common and, from the standpoint of public health as well as the dairy industry, probably the most important. For these reasons and because of time limitation, the discussion will be limited to that subject.

The most common incitants of the condition we often speak of as "ordinary" bovine mastitis are streptococci which, for a long time, have been classified in a group given the name *Streptococcus agalactiae* or *Streptococcus mastitidis*. These, for a long time, were looked upon as non-pathogenic for man but, as studies of streptococci have progressed, evidence has been accumulating that indicates that this was an incorrect assumption. While they seldom cause the most active and acute human infections, streptococci falling into this general group have been found to have been associated with a variety of human infections. For example, they have been isolated from gastric ulcers in a series of cases and from fatal cases of uterine infection.

### LANCEFIELD'S CLASSIFICATION

At this point, I want to stop and go into a little detail concerning this matter of classification of streptococci and the association of those of certain classes with bovine and human infections. I do it with the reservation that I am not a bacteriologist and can give you only my understanding of the facts. The changes from old to new classifications are confusing and my only hope is that I will not add to the confusion. However, it is necessary to get some of these things straight in order to appreciate the relationship between the bovine and human infections. This we must do, naturally, if we are to understand the relation between mastitis and public health.

The latest and most generally accepted classification into Alpha, Beta and Gamma streptococci is based on the reactions produced by the organisms on blood-agar plates. Those of the Alpha group, which includes what we have called *Streptococcus viridans*, produce a brownish-green discoloration and only slight or incomplete hemolysis of the blood-cells in the medium. We can disregard this group, as far as this discussion is concerned. Those of the Beta group produce clear, colorless zones of hemolysis, and hence are spoken of as *Beta-hemolytic* streptococci. This is the group that particularly concerns us. Those of the Gamma group produce no hemolysis.

Lancefield, on the basis of serological tests, has subdivided the Beta-hemolytic

*Presented at the seventy-fifth annual meeting of the American Veterinary Medical Association, New York, N. Y., July 5-9, 1938.

streptococci into nine sub-groups, which she has designated A, B, C, D, E, F, G, H and K. This is called Lancefield's classification and appears to be quite generally accepted.

Group A is the most important, from my present standpoint, because it consists almost wholly of Beta-hemolytic streptococci which are exclusively of human origin. We shall have more to say about them presently, because they are responsible for the cases of mastitis to which milk-borne outbreaks of septic sore throat and scarlet fever are traced. Group B probably corresponds roughly to the old agalactiae grouping, because it is this group which is said to be the chief cause of bovine mastitis but which, as already indicated, has also been isolated from a variety of human infections. We are particularly concerned, then, with Beta-hemolytic streptococci of two of Lancefield's sub-groups: A, of human origin, but which will incite mastitis in cows and is responsible for milk-borne epidemics and B, which is the common incitant of bovine mastitis, but is associated with certain infections in man.

When thinking of these streptococci as being divided into different groups, there are two things to be kept in mind. One is that demarcations between groups of bacteria are not sharply defined. There may be borderline strains. Another is that some of these organisms, under conditions which can not be predicted, may take on new characteristics. When practicing medicine, back about 1907, we did not think of poliomyelitis (infantile paralysis) as a communicable disease. When we had an occasional case, no precautions were taken. Yet, in 1916, this disease spread over the country like wildfire. For many years after the pandemic of influenza in the 80's, cases and outbreaks occurred here and there, but, in general, it was not a serious matter. Then, when men of military age were gathering in camps for the World War, the virus, apparently as a result of having been passed rapidly from man to man, took on a new virulence, particularly for men of that age. So there is always the possibility that strains of "mastitis streptococci" of group B, under just the right set of conditions, may assume a new virulence for man. Certainly we are no longer safe in saying, as we used to, that these organisms are "harmless for man."

Now we come to what, to me, is the most interesting part of the subject, namely, udder infections due to Beta-hemolytic streptococci of human origin, group A under Lancefield's classification. These are the organisms responsible for scarlet fever and septic sore throat. They, also, seem to be highly infective for the bovine udder, when there are breaks in the protective tissues through which they can enter. As far as we know, accidental infections with these organisms occur as readily in normal udders as in those previously infected. I mention this particularly because the fact that mastitis in general is less common in the southern states has been cited as a possible reason why some of these states discover no milk-borne epidemics, or very few, traceable to this type of udder infection. I doubt if this is the explanation.

We have milk-borne outbreaks of septic sore throat or scarlet fever quite regularly in this state—all of them, however, "upstate," as we say,—because here in New York City, where practically all of the milk is pasteurized, we apparently do not have them. We find some compensation for the disgrace of having these epidemics in the satisfaction arising from the belief that we are now discovering and tracing most of them to their original sources. From this study we have obtained information of importance. The states that are letting the milk-borne epidemics of these diseases go undiscovered, I have no hesitation in saying, are missing a lot of fun, to say nothing of the opportunity to do something for public health.

Our experience and that of others have led us to several important conclusions. One of these is that nearly all extensive milk-borne epidemics, of septic sore throat, and many lesser ones, together with many of scarlet fever, are traceable to milk from cows with mastitis. Nowadays, when we

fail to locate a cow, we suspect that something has been overlooked. However, our veterinarian-epidemiologist, Dr. Graves, has developed such an eagle eye, figuratively speaking, that he seldom misses the cow if he gets on the job before she has been sent to the abattoir. Of course, we never conclude that the accountable cow has been found until his observations have been confirmed by laboratory findings.

Another very definite conclusion is that, whenever a case of mastitis is responsible for such an epidemic, the cow's udder invariably has been infected with Beta-hemolytic streptococci from a human source. Usually it is a milker who has a throat infection or, less frequently, a wound infection. The ordinary "mastitis strep," Lancefield's group B, while it causes most of the mastitis and apparently may at times infect man, as far as we know never causes septic sore throat or scarlet fever.

### DANGEROUS CARRIERS

During the past two years we have had three milk-borne outbreaks of streptococcus infection in which the cows and the human sources of their udder infections have been located, and in which the infection of the cow's udder, in each instance, occurred in one place and the outbreak in another. I want to tell you about the least spectacular of these because it had one significant feature not present in the other two, and also because I am able to show you a very striking picture of a microscopic section of the cow's udder.

We refer to this cow as the "Tyrone cow" because we first made her acquaintance in a town by that name. In July, 1936, 16 cases of scarlet fever occurred in the town. They were definitely traced to this cow. She had a mastitis, from which Beta-hemolytic streptococci of group A, corresponding with those on patient cultures, were isolated. It was then found that, in May (the outbreak was in July), she was milked by a person who, from the description, very evidently had mild scarlet fever. Then, something unforeseen happened. The dairyman assured our district milk sanitarian that he would either slaughter the cow or sell her for slaughter. Ordinarily, our man would have followed this up, but he was suddenly stricken with pneumonia and forget all about cows for several weeks, and about this particular cow for eight months. Then, one day, when asked what happened to her, and being unable to answer, he went to Tyrone and learned that the original owner had sold her but did not know her fate. It developed that she had changed hands several times. Our man traced her from farm to farm until he came to one in the town of Starkey, in another county. Among the first things he noticed were scarlet fever quarantine

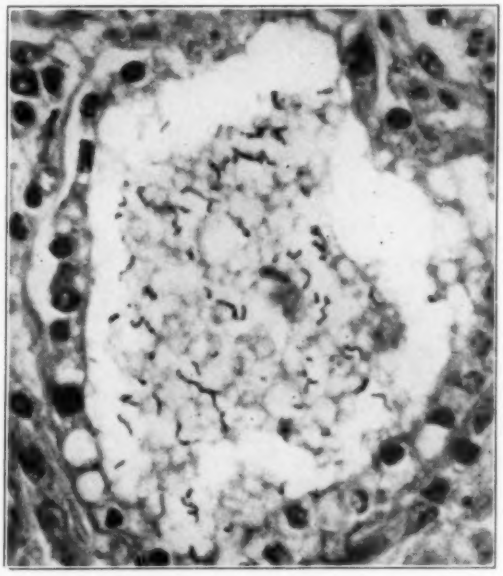

FIG. 1. Photomicrograph of section of udder of cow responsible for two milk-borne outbreaks of scarlet fever, six months apart (x 650).

signs on the farm and tenant houses. There were nine cases on the farm. All of this cow's milk was consumed on the farm and all who had used it had scarlet fever. She had freshened shortly after arriving at this farm and our man again found her to have a well-developed and obvious mastitis.

In short, following the first outbreak and during the dry period, the streptococcus infection had remained latent, lighting up after the next freshening. At least one investigator had previously observed and reported that mastitis, incited by artificial inoculation, subsided when the animal

"dried off," and recurred later when she freshened again. This was the first reported instance, as far as we have been able to determine, of a recurrence, under natural conditions, resulting in a second epidemic. At least we learned something from our oversight.

This time there was no question about her disposition and her udder is now an interesting specimen in the museum of the state laboratory. The accompanying photomicrographs (figs. 1 and 2) taken at two

FIG. 2. Photomicrograph of section of udder of cow responsible for two milk-borne outbreaks of scarlet fever, six months apart (x 2,000).

different magnifications show the streptococci, presumably of the same strain that caused the outbreaks, standing out prominently in the milk-cisterns and canals.*

Dr. Graves now has detailed notes of the results of his examinations of a number of these "epidemic" cows. I hope he will find time, before long, to analyze and write them up for publication. In the meantime, I can give only my impression, based on second-hand observation over 15 years or more, that it is impossible to say, from physical examination of an inflamed udder, whether the mastitis is one of the dangerous kind or relatively harmless. Furthermore, an epidemic may result from a case in a stage so early that it might be missed with any method other than a thorough examination.

The only way they can be differentiated is by time-consuming laboratory tests of a sort not suited to routine examinations and practically never made except experimentally or when an epidemic occurs. This being so, every case must be regarded as potentially dangerous. It would be significant if it could be determined that the cow had been milked by an infected person, but I have never heard of this being done in the absence of an epidemic.

Finally, we come to the question as to what should be done about it. To those of us who are officially responsible for the protection of public health, our practical and immediate concern lies in preventing cases of mastitis from becoming sources of human infection. How can this be done? Many of you, I think, will be able to anticipate my answer.

Except in isolated and special instances, it can not be done by veterinary inspection of herds. In certified dairies, where the requirements as to veterinary examinations are rigidly and conscientiously observed, there is no doubt that these examinations afford a good deal of protection. Yet, a few years ago, we had an outbreak of septic sore throat on a government reservation, clearly traceable to certified milk. In the herd, which was reported to have been examined by a veterinarian shortly before, a well-marked case of mastitis was found. Due to complications arising from the fact that the epidemic was on a government reservation, our investigation was never completed. A few years ago, on the occasion of a milk inspectors' meeting, in another state, I went with a group to visit a certified dairy considered a "show place." We observed an obvious case in the milking line. One of us asked the stableman if they had noticed it and he said: "Oh, yes. We're going to take her out later." The cow accountable for the largest of the three epidemics mentioned earlier in the paper was reported to have been examined, with others, a few days before the epidemic and

*Photomicrographs taken by Dr. Schleifstein, of the New York State Laboratory.

certified to be sound, although the owner admitted knowing at the time that she had mastitis. Probably these are not fair samples of veterinary examinations, but veterinarians vary as to their honesty and efficiency, just as physicians do. We have to depend on "the run of the mine." Moreover, the veterinarian can not spend all of his time at one dairy.

Much might be accomplished if dairymen would keep persons whom they know, or should know, to be infected away from their cows. But dairymen, generally speaking, won't—not in the in the present state of sentiment and enlightenment. Regardless of the danger to their cows, persons with sore throat and other obvious illness should not be milking. Yet, over and over, investigations of epidemics reveal that this has been done. Our regulations provide that milk from cows with mastitis shall be immediately excluded from that to be sold, yet, again and again, we find that it has been knowingly included.

### MASTITIS CONTROL AN ECONOMIC MEASURE

There has been a great deal of discussion in this state and, I suppose, in others as to whether a campaign for the general eradication of mastitis is a public health measure. I have been severely criticized once or twice for saying that it is primarily an economic measure for the farmer, and only incidentally a public health measure. Any material reduction in the amount of mastitis will improve the quality of our milk supply and increase both its healthfulness and its marketability. At the present time a very few intelligent and progressive dairymen, by application of scientific control measures, are keeping mastitis in their herds down to a minimum. I would not be surprised to see, in the next few years, some general appreciation of the economic importance, to the individual farmer, of reduction in mastitis in his herd.

But even if the general run of mastitis could be eradicated—something I am afraid none of us will live to see—it is quite unlikely that it would prevent the occurrence of the cases of mastitis responsible for our scarlet fever and septic sore throat epidemics. Why? Because milkers still will have streptococcic throat and wound infections and they will continue to infect udders, regardless of whether the cases of "ordinary mastitis," as we call it, are few or many. Don't misunderstand me. I am in favor of all reasonable and practical efforts to reduce the amount of mastitis. It will benefit the farmer principally and the public health incidentally. But as a measure for the prevention of milk-borne epidemics, its value will be practically nil. This leaves only one readily available and practically certain means of protecting the public—and that is pasteurization.

Perhaps it is not too much to hope that in time—not a little time but in the course of a long time—a majority of dairymen will have learned to keep persons with obvious infections away from their cows and away from the milk; perhaps they will even learn to protect the udders of their cows from infection and reinfection with the now so common "bovine strep." But, probably there still will be the human carriers whose infections are not obvious—carriers of the organisms of diseases like typhoid fever and even, possibly, of diseases not now regarded as milk-borne. We still will have the minority; less intelligent and less efficient. We still will have mastitis and milk potentially dangerous. As far as the public health is concerned, we will have to depend on pasteurization, unless, in the meantime, someone devises a better method for destroying pathogenic organisms.

### DISCUSSION

DR. F. D. HOLFORD: I would like to ask Dr. Brooks if, in a program of mastitis control, a dairyman who is interested in controlling his herd, even if he did have a case of mastitis from human infection, would be better benefited if his milk were sold raw, in view of the danger of an epidemic of septic sore throat, because as soon as he found that cow having mastitis he would eliminate the milk from the supply, and therefore the mastitis-minded farmer, so to speak, would have a big public health significance.

DR. BROOKS: I thank Dr. Holford for having answered his own question. The answer is "yes."

DR. F. W. GRAVES: There are several important factors I should like to stress. I am a veterinarian, employed by the New York

State Department of Health as a milk sanitarian, doing general field work and assisting our Department of Communicable Diseases in the investigation of milk-borne diseases.

It is a big field, one in which we need more veterinarians who should become interested in the field of public health. I should like to create in the minds of you veterinarians a desire to get into this field of public health much further than you have gone. As Dr. Brooks said, and as Dr. Holford and many of the men who have worked on this can verify, there is a sporting side to it which surely needs your consideration in the field of veterinary medicine. The investigation of epidemics is really sporty, because you don't know where you are going, although you do know what is expected of you. The epidemiologist has told me there is an epidemic present and that, from the indications, it is milk-borne and, when epidemics are milk-borne, especially scarlet fever and septic sore throat, a cow is usually the cause and our duty as veterinarians is to find the cow.

Epidemics—we have had a lot of them in this state, and I am speaking primarily of septic sore throat and scarlet fever, gastroenteritis and undulant fever. Since 1930, to give you a few of the major ones, we have had epidemics at Walton, Greenwich, Norwood, Luzerne, Baldwinsville, Hicksville, Wayland, Cobleskill, Red Creek, Waterloo, Owego, and in other villages I can not recall just now. They have all had interesting features; they have all been cases where we have been pretty well able to definitely pin down the source of the trouble.

The subject of mastitis has two phases. You men as veterinarians have to approach it from the economic and public health side. You have been approaching it primarily from the economic phase. Maybe that is proper; maybe I have gone a little bit prejudiced. However, I believe you men as practitioners (and I had been a practitioner for 16½ years before I entered the public health work) should look at the public health phase of this matter. You have a chance to initiate work that has never been accomplished or attempted on a very large scale.

Every one of you has similar problems in your own community. I have had the pleasure, during the last year, to work on several certified farms with Dr. Hopson from the Kings County Certified Milk Commission. I have had the pleasure of being sent into herds under the control of Dr. Holford's staff. I have had the pleasure of being in herds under the veterinarians supervised by Dr. Corbin, and I have seen some very varied conditions. I have also seen some of the herds which have had practically no veterinary supervision. The public health protection to a raw milk supply has not given any greater protection to a community with the well organized plan of veterinary supervision than is afforded by the herd with no veterinary supervision. Even though mastitis should be eliminated from the herds for both public health and herd economy, pasteurization is our only dependable safeguard to a community milk supply.

This last winter, we had one herd in a little suspected epidemic, in which we were not able to prove it as septic sore throat, in which only 50 or 60 cows were involved. As I approach a herd examination in epidemics, I classify it as A, B and C, trying to follow the Lancefield type of classification. My type A cows are cows that cause these epidemics; the type B cows are the ones that have hemolytic streptococcus not of the human origin, and under this class may be included streptococci or other organisms that may produce toxic gastroenteritis. The class C type includes the other types of mild mastitis which are destructive economically to the herd, which do not produce epidemics and are not serious as a public health problem, but this type of mastitis does injure the quality of the product.

Out of some 42 herds we examined in the epidemic Dr. Holford spoke of in the *Health News*, and in examining something like 1,000 cows, we picked out the cow that caused the epidemic. There were less cases of mastitis in this group of 42 herds of the type B classification than there were in this little epidemic in which only five herds were involved.

Why? Because these 42 herds, out of a total of 199 herds producing milk for the plant involved, had been under good veterinary supervision. The owners had been worked with for several years; the dairyman knew what the responsibility would be if they went out and purchased cows from cowdealers and brought disease into their herds. They practiced good dairymanship.

One other matter that I would like to stress for the benefit of the veterinarians, speaking as one myself, is that I am afraid if we as veterinarians do not bear down and do more intensive work on mastitis and become more interested in that subject, and assume our responsibilities in the public health field, the engineers and the bacteriologists, for whom I have the greatest respect, are going to come along and take work away from us which belongs in our field. Veterinary examination of herds is generally of the poorest quality, except in Grade A dairies operated by some of the large milk companies. I know of a certain group of health officials right now who are attempting to promulgate the idea that the proper control of a milk supply is the laboratory, by the taking of Breed smears or direct microscopic examination, having the result of these Breed smears followed up by lay inspectors, who go out to the farm and take individual quarter samples from the cows, segregate the suspected animal (without legal authority) and leave the veterinarian out of the picture. That type of supervision is gaining headway, and we as veterinarians are the direct cause of it, because of poor veterinary examinations and indifference toward our responsibility to good milk production. I am giving you veterinarians, particularly those of New York state, this advance information and you can take it for what it is worth. If we don't bustle into it and do our jobs, we are going to suffer and lose our place in community welfare in the field of public health.

May I stress again the remarks that Dr. Hardenbergh made earlier relative to the mastitis demonstration downstairs. To me it is very interesting and, most surely, should merit the attention of all practicing veterinarians. I obtained the mimeographed copy describing the demonstration this morning and read it over. I hope to read it over again tonight, and then, tomorrow, I shall study that demonstration more thoroughly. It is practical and has unlimited value to veterinarians who may be called upon to conduct physical examinations of dairy cattle.

May I suggest, Dr. Hardenbergh, that all veterinarians who are interested in mastitis get that copy and study it, and then go down tomorrow and study the exhibit, study the graphs, the plates, the histories they have there, and see if we cannot create more interest among ourselves relative to more efficient examinations of udders. May I again stress the fact that we as veterinarians are going to suffer almost irreparable loss of respect and prestige in the field of public health and as professional advisors to our clients, unless we assume our place in the field of preventable, communicable diseases of man and our domesticated animals.

## Meat Inspection Costs Low, U. S. Records Show

Veterinary inspectors of the United States meat inspection service often are asked, "Who pays for meat inspection?"

Federal meat inspection is paid for by the government—not by the packers nor by the meat trade. Official records show that the total cost of all operations, including administration, is about 7 cents for each animal slaughtered. This is about 1/50 of a cent a pound for all meat products inspected or between 3 and 4 cents a person a year.

There are other benefits besides protection to human health. The Bureau of Animal Industry, which conducts the meat inspection service in all establishments engaged in interstate commerce, generally is able to trace shipments of diseased cattle to their source. Farmers, ranchmen, and officials engaged in disease-eradication projects appreciate this service. It helps to locate and remove causes of infection, improve farm conditions and, in turn, cut production costs.

Another question often asked regarding federal meat inspection is, "What is done with condemned meat and meat products?" All condemned meat is constantly in charge of inspectors until it is destroyed for food purposes, as by reduction to fertilizer and inedible grease.

## Doctor Carpenter Now a Hoosier

Dr. Clifford D. Carpenter, who was engaged in research work at the University of Kentucky the past year, is now with the Allied Mills, Inc., of Fort Wayne, Ind., as pathologist in the Educational Department of the company. In his new work, he will be closely associated with a number of well-known authorities on nutrition.

DR. C. D. CARPENTER

Since his graduation from Cornell University in 1920, Dr. Carpenter spent three years at the University of California Experiment Station, and then resigned to establish the first private laboratory in the United States devoted exclusively to the control of poultry diseases. In 1935, he became associated with the Lederle Laboratories, in New York City, as poultry pathologist.

## Rare Horse Disease

Equine encephalomyelitis got more than its share of newspaper publicity throughout the country during the late summer and fall months. Some of the rural publications had considerable difficulty with the nomenclature of the disease. The prize, however, goes to the Lebanon (Ky.) *Enterprise*, which announced "Rare Horse Disease in County," under date of November 4, 1938. The article reported that "encephalopyosis" had appeared in the county.

# The Physiological Background of Transfusion Therapy*

*By* ROGER S. AMADON, *Philadelphia, Pa.*
*School of Veterinary Medicine, University of Pennsylvania*

During the past few years, transfusion therapy has become the object of a greater amount of interest and attention in veterinary practice than ever before. The value of this procedure in the treatment of blood-vascular disturbances has been fully appreciated by the profession in previous years, but freedom of application has been restricted by numerous handicaps peculiar to the field of veterinary practice.

The writer has been greatly interested, during the past ten years, in the question of perfecting methods that would enable the practitioner to apply transfusion more frequently in routine practice. An effort has been made to clarify questions pertaining to indications, contraindications, methods and equipment necessary to the successful utilization of this valuable therapeutic measure by the veterinarian. As indicated by the title, this paper will stress in particular the more important facts relative to the physiological background of transfusion therapy.

The fact that transfusion is referred to as a therapeutic measure implies that some disturbance of body function has developed which should be benefited by the intravenous injection of a quantity of blood or of saline solution. To appreciate fully the therapeutic value of the measure and to be enabled to judge more accurately the extent of its application, it becomes desirable that a certain amount of attention be devoted to a brief review of the physiology of the blood and circulatory system.

The subject matter of physiology represents the effort of man to explain the phenomena of functional activity as it occurs in the normal animal or plant body. This activity occurs with a certain degree of intensity and fluctuates between certain limits in consequence of regulatory control. Though absolutely essential to all effort that may be directed to the prevention and cure of disease, physiological knowledge has failed, nevertheless, to receive the attention lavished upon pathology by the medical public.

The unexpected and often little understood phenomena that constitute pathology have a greater power of attraction for the human mind than is true of the routine of regulated phenomena persisting throughout the normal life of an animal body. However, the fact remains that any effort to understand cause and effect operating to produce perversion of body function is fruitless in the absence of physiological knowledge and is just another example of a reversal of cart and horse.

## BLOOD-VASCULAR PHYSIOLOGY

Directing attention to the physiology of the blood vascular system, it may first be stated that this term is used to designate the heart, blood-vessels and blood as a unit. In brief, the work of this system consists of providing transport facilities for the vast community of tissue cells that constitute the mammalian type of animal body. The analogy of the body to a community is entirely proper, with few restrictions. It is an ideal community, free of politics, and with coöperative effort exemplified to the greatest degree possible. The tissue cells, fixed as they are in location (with the exception of those entering into the composition of the blood and lymph), are wholly dependent upon the blood-vascular system for the receipt of material required in the course of their metabolism and for the removal of metabolic wastes.

In consequence of the vitally important contribution to the functional activity of the body, the clinician must devote particu-

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lar attention to the working efficiency of this system. In the treatment of practically all disease conditions, the functional activity of the blood and circulatory system receives a certain amount of consideration. Among the materials delivered in the tissues, oxygen is of chief importance as regards the maintenance of life. Mammalian tissues are incapable of storing any appreciable amount of oxygen, so that the blood-stream must serve as the ever-ready and constant source of supply. Oxygen deficiency is productive of a very prompt disturbance of function in nervous, epithelial and muscular tissue which, after an interval of minutes, will terminate fatally if the supply fails completely.

Blood is admirably adapted to the task of oxygen transport by virtue of its content of the respiratory pigment, hemoglobin, which is distributed over the vast surface constituting the structure of the red corpuscles and is thus freely exposed to body environment for the taking up or releasing of oxygen. As the blood flows through the tissue areas, it releases only a portion of its oxygen content, some 25 to 85 per cent being present after the tissue requirements of working and resting conditions have been satisfied. This excess of oxygen-carrying power possessed by the blood is one of a considerable number of instances of surplus or reserve function provided by nature as a safeguard to tissue health and life-body resistance to disease-producing agencies. It depends, in large part, upon these reserves.

In evaluating the quality of blood, the principal factor is hemoglobin content. To a lesser degree, the question of quality is dependent upon the composition of the plasma or fluid fraction of blood which carries many inorganic and organic substances, in a state of true or colloidal solution. Water constitutes 80 per cent of the blood. It serves as the solvent for plasma ingredients, is essential to the functional activity of blood-cells, and finally it provides the proper consistency required for the free flow of blood through the cardiovascular circuit. Blood analytical methods today are affording the clinician valuable aid in this matter of ascertaining the quality value which serves as guidance in diagnosis and treatment of disease.

The heart and blood vessels, with their nervous regulation, constitute the means for guiding a steady flow of blood into all parts of the body. The heart constitutes the power plant for the system which provides the energy required to drive the blood out through the main and branch lines of the arterial part of the circuit and along the countless, narrow, capillary channels. A special type of muscular tissue is responsible for the energy supply, energy that must be available at but a few seconds interval from early intra-uterine life until the termination of body existence. The propulsive power of heart-muscle is instrumental in providing a rich blood-supply which this vital, hard-working tissue requires. The coronary arteries carry a greater quantity of blood when the work of the heart muscle is increased by cardiac acceleration or by any increase in the level of aortic pressure, the increased flow being due to the fact that the coronary arteries originate from the initial part of the aorta, where systolic pressure reaches its maximum level.

Under physiological conditions, this relation of blood-supply to cardiac function operates to excellent advantage, but when the heart action becomes weakened by disease a very serious state of disturbed balance frequently becomes established, as will be discussed later in this paper. Muscle tissue requires a certain amount of rest in order to recover from the changes produced during the contraction period. Heart muscle recuperates during the pause intervals of the cardiac cycle, so the fewer cycles per minute the more extensive will be the resting period. Within a rather extensive physiological range of frequency, the work of the heart is performed efficiently without fatigue or injury. Beyond a certain frequency, however, reduction in volume discharge occurs. This condition may be observed when the venous return to the heart has been slowed. Such a state of

affairs imposes a double handicap upon the heart, as both its blood-supply and rest periods are reduced.

The physiology of blood-vessels involves functional activity of arterial, capillary and venous vessels. The arteries possess strength to store a relatively high level of energy as provided by the force of heart action upon the column of arterial blood. The intermittent fluctuations in blood-pressure at the cardiac end of the arterial path change to a steady flow from the terminal arterial channels into the capillaries. All possibility of cessation of flow during the pause period of the cardiac cycle is thus eliminated. The nonstriated muscle present in the arterial walls serves to modify the caliber of these vessels and is of importance in shifting blood from inactive to active tissue areas, in counteracting the distribution disturbing effect of gravity, and in adjusting the capacity of the arterial system to blood-volume so that a constant level of blood-pressure will be maintained. A nervous regulation exercises vasodilator and vasconstrictor control over the muscle portion of the arterial structure. The head of pressure existing in the arteries is utilized for the driving of blood through the great expanse of capillary vessels, where resistance to flow is very great.

Only a small residue of blood-pressure persists in the capillary channels. The capillary portion of the vascular system is the place where the blood-stream is unloaded of a part of its content of nutritive material and is loaded with waste products of tissue metabolism. Structurally, the capillary walls are extremely thin and well adapted for the exchange reactions occurring between blood and tissues. The microscopic dimensions of these vessels serve to reduce the blood-stream to the diameter of one or two erythrocytes, thus exposing both cells and plasma to the maximum effect of filtration, osmosis and other agencies responsible for the exchange process.

The collection and return of blood to the heart is accomplished by the veins. Only a very minute residue of the propelling force of the heart remains at the venous side of the capillaries and contributes very little to the flow of blood in the veins. Because of its relatively thin walled structure, the effect of forces acting external to the veins is transmitted to the blood-content of these vessels. Striated muscle functioning in the course of locomotion and respiration provides the major amount of energy for the venous circulation. Veins are equipped with valvular structures which prevent any back flow in the direction of the capillaries.

Respiratory movements are a very important aid to the venous circulation, as the alternate increase and decrease in thoracic and abdominal pressure has an aspirating and compressing effect upon the great veins of the trunk region. These movements of respiration also are operating during periods of locomotor inactivity. The efficiency of the circulation is seriously disturbed by a reduction of the venous flow to the heart. Under normal conditions, a sufficient amount of blood is delivered to the right side of the heart to meet all of the requirements for blood-supply to tissue in a state of rest or work. Disease, however, may cause a disturbed balance of venous return and systemic demand for blood, as will be discussed shortly.

This abbreviated, probably irksome, résumé of certain phases of the functional activity of the blood-vascular system will assist in understanding more clearly the relationship of transfusion therapy to certain diseases of this system.

### HEMORRHAGE AND SURGICAL SHOCK

Blood-vascular disease produces a syndrome of symptoms that may be the result of primary or secondary changes in the quantity and quality of blood and in the normal circulation throughout the body. The question of quantity will be considered first.

Quantity is modified clinically by hemorrhage or surgical shock in the direction of a reduction in the volume of the total blood or of its moisture content. Hemorrhage from any cause reduces the volume of blood and, if it occurs to an extensive degree, it will incapacitate the blood-vascular system

to a fatal degree. Slow, prolonged loss of blood is sustained much better than a rapid loss of smaller quantities. This is explainable in terms of vasomotor compensation and moisture retention by the blood-stream.

Vasomotor nervous control of the arterial side of the circulation in particular serves to bring about a constriction of the vascular channels and, in consequence, a reduction in capacity to accommodate the lessened volume of blood. Blood-pressure remains undisturbed within certain limits in slow hemorrhage. In addition to vascular adjustment, the heart rate also is accelerated by its nervous regulation. The mechanisms regulating the heart and blood-vessels are afforded an opportunity to keep pace in their action with the diminishing of blood-volume, and a third or even a half of the total blood may be lost and recovery still occur. Rapid blood loss is accompanied by an immediate decline in blood-pressure, which slows or completely checks the capillary circulation, and tissue functions are depressed in all portions of the body.

Death occurs, due to asphyxial failure of respiratory and vasomotor centers and the heart. In the event of a non-fatal hemorrhage, a recovery of normal volume occurs within 24 to 48 hours, the return to normal being accomplished at the expense of the general store of tissue fluids, assisted by increased water intake caused by the stimulation of the thirst sense. During the period of volume recovery, the blood-count, hemoglobin concentration and cell volume will decline progressively in consequence of the blood dilution occurring and will remain at a level below normal until a return to normal quality has been effected by the blood-forming tissues.

The process of restoring the total blood-volume to n o r m a l is of much greater importance than the recovery of normal composition, as a good circulation of blood of inferior quality will serve at least to sustain life, while defective circulation of blood of inferior quality causes serious disturbance or even death. This fact is exemplified in many chronic diseases that produce severe anemia, in the presence of which life processes are being carried on successfully, although within a restricted physiological range. The great reserve of oxygen-carrying power of the blood accounts for the failure of quality reduction to bring about immediately fatal results.

Surgical shock can be discussed advantageously in connection with hemorrhage, as it presents a symptomatology very similar to that of severe hemorrhage. The character of the disturbance consists of volume reduction in the cardio-arterial part of the circulatory system with a capillary venous stasis and engorgement. Examination of blood in shock reveals a marked increase in cell volume, indicating that dehydration has occurred and, in advanced stages, the volume relations of cells and plasma will be found to be reversed. The significant feature of moisture loss from the blood-stream is the great increase in viscosity which retards the normal flow. Moisture can escape from the vascular system into the tissue spaces only in the capillary regions and an abnormal freedom of escape must occur in connection with the development of shock.

Injury to the delicate structure of the capillaries is productive of a greater freedom of filtration from the blood-stream and a source of such injury apparently becomes established in surgical shock. As the condition follows in the wake of extensive tissue damage inflicted by the surgeon, trauma or severe inflammatory reaction of mucous and serous membranes, it is logical to suspect that this may serve to release toxic substances into the blood-stream. Experimental observations lend strong support to this view, but the specific character of the toxin or toxins still remains to be ascertained. Judging by the ability of histamine, a derivative of the amino-acid histidine, to produce a quite typical shock state, it may be possible that cytolytic changes developing in crushed tissues result in the formation of histamine or closely related compounds.

The disturbance of circulation apparently is initiated by toxic damage to the capillary walls, resulting in an increase in the filtra-

tion rate. As the consistency of the blood becomes less fluid, the transfer in the capillaries and veins is accomplished with increasing difficulty, and stasis develops with a reduction in the return of blood to the right auricle. Capillary and venous stagnation is favored, due to the absence of the driving force of the heart in this portion of the circulation. In the absence of a normal volume of return flow, the heart is incapacitated in its work as a pumping mechanism and general blood-pressure eventually declines to subnormal levels.

A low blood-pressure stimulates the regulatory control of heart and blood-vessels, cardiac acceleration and vasoconstriction becoming established in a fruitless effort to build up a higher level of blood-pressure. With this picture in mind, the clinician can readily appreciate the gravity of the situation and the necessity for guarding against the establishment of such a vicious cycle. The condition, when once well established, progressively becomes worse until the heart finally fails, due to an insufficient blood-supply and an accelerated effort.

The treatment of hemorrhage of a severe degree and of surgical shock is directed towards a prompt return to normal of the volume of fluid in the circulation. Blood transfusion is specifically indicated for both conditions as a life-saving measure of supreme value.

Compared with the use of blood, saline transfusion is of less value in the patient suffering from hemorrhage and of no value in the animal suffering from shock. In the treatment of hemorrhage, saline transfusion will prove of value as an emergency means of preserving life when the limit of blood has been reached and a transfusion of blood is not possible, due to the absence of a donor. Blood-pressure may be restored under these conditions, but the recovery is often temporary, due to the escape of transfused saline by capillary filtration. Blood gives more permanent results in consequence of the content of protein which is retained in the circulatory channels and counteracts capillary filtration by providing a low, but constant, osmotic tension.

In addition to providing for the correction of volume deficiency, the use of blood is of valuable assistance in restoring hemoglobin concentration, which is very desirable, even though the hemorrhage patient is robust and can be relied upon to recover eventually unassisted. Hemoglobin is not only contributed to the patient's bloodstream, but a stimulus to increased activity on the part of the hematopoietic tissues is reported to occur.

The treatment of well established conditions of surgical shock is very rarely attended with success. It is a disease condition that must be prevented, if possible, or at least subjected to treatment early in its course of development. The inability of veterinary patients to receive close attention and constant care afforded in the medical care of man readily leads to the conclusion that the veterinarian is confronted with hopelessly advanced shock conditions and such a conclusion is unquestionably true. The term secondary shock is often used to designate this particular disease because of the relatively long interval between the cause and effect. The circulatory upset is effected several hours after the surgical operation, the accidental injury or any other condition being associated with a sufficient amount of tissue injury to set up a shock state.

The diagnosis should be based upon the history and an examination of pulse and, if possible, a centrifuged sample of blood. As few practitioners have access to a centrifuge, it might be suggested that a rough estimate of increased cell volume may be made by noting the freedom of flow from a bleeding-needle. The insidious development, usually at a time when the patient is unobserved, and failure of early diagnosis serve to favor well advanced and fatal conditions of shock in veterinary patients.

Any attempt at treatment should be concerned with an effort to introduce a sufficient volume of fluid into the arterial circulation to elevate blood-pressure to a point where the capillary stasis is removed. The principal difficulties in accomplishing this objective are, first, the ability of a weak-

ened heart muscle to drive blood into the arteries to the extent of developing an effective level of pressure. Second is the question of maintaining the increased volume within the vascular circuit in the presence of capillary damage.

Blood, transfused at a slow rate, is the only therapeutic measure that will satisfy requirements. Injection must be performed very cautiously, the pulse being observed constantly. Recovery of cardiac muscle cannot be expected to occur within a brief interval of time and any improvement in strength of pulse marks a returning blood-supply to the heart, which is a step in the right direction but should not create a desire to hasten the transfusion. The heart must provide the muscular energy for its own recovery. The weakened cardiac muscular tissue may be overpowered by too rapid a demand for functional activity. Judgment must always be exercised in the intravenous introduction of any fluid. The common error consists of speeding the administration too greatly and failure to allow body tissue time enough for adjustment to the new conditions established. Particular attention is necessary in the presence of a heart that has been suffering for a variable period of time from deficient oxygen and nutritive supply. Saline solution, as previously mentioned, fails to give satisfactory results in the shock patient, due to the very rapid loss from the circulation through the injured capillary walls.

### TRANSFUSION IN DISEASES MODIFYING QUALITY OF BLOOD

Certain disease conditions produce marked disturbance in the quality of the patient's blood, such as sweet clover poisoning of cattle, purpura hemorrhagica of the horse, and the anemias and hemophilia-like conditions appearing in any of the domestic species. The quality changes that occur in these diseases are all marked by disturbance of blood-vascular functions and thus are capable of fatal injury.

In sweet clover poisoning of cattle, the disturbance of physiological reaction consists of a loss of the coagulating power of the blood, partially or entirely, and is accompanied by toxic injury to the vascular system, so that extensive and fatal loss of blood by internal bleeding occurs, or by external hemorrhage from surgical or traumatic injury of blood-vessels.

The particular defect in the coagulation reaction has not been revealed by experimental studies. Transfusion of 500 to 1000 cc of normal blood into the patient is very effective in restoring the normal coagulability of the blood and bringing about a recovery from the disturbance. At present, there is no answer to the question of what particular reaction is brought about in the patient's body that is responsible for the beneficial effect. Blood, when removed from the circulation, and especially after the coagulation reaction has been completed, as in defibrinated blood, possesses vasoconstrictor properties and hastens coagulation.

Purpura hemorrhagica presents the symptomatology of a severe capillary intoxication associated with changes in the composition and functions of the blood. Extensive leakage of plasma and blood-cells which occurs in capillary regions is indicative of serious injury to the endothelium of these vessels. The writer has felt assured, for the past few years, that the introduction of fresh normal blood by transfusion would be of value. A number of practitioners have put this opinion to test and have reported very gratifying results. Several small transfusions of 300 to 500 cc of blood are usually required in the treatment of severe cases. In some respects, purpura resembles the above-mentioned clover poisoning of cattle, but the disturbance of coagulation is much less pronounced; at least, it is not stressed by authorities in veterinary medicine. Again, the vasoconstrictor effect and the acceleration of coagulation time apparently act to good advantage in retaining a normal relationship between the volume of tissue fluid and the volume of the blood-stream.

Anemia represents a distinct decrease in the functional value of the blood as an oxygen-transportation agency. The addition of fresh blood to the circulation is clearly indicated, often is of permanent value and always produces temporary im-

provement. If disease agencies have incapacitated the blood-forming tissues of the body or destructive agencies are eliminating large numbers of blood-cells, repeated transfusions must be performed to maintain life and a reasonable degree of health.

Transfusion not only provides a fresh supply of erythrocytes, but also is capable of accelerating the production of new cells by a stimulant action on the bone-marrow. The patient convalescing from debilitating disease conditions is commonly found to be low in hemoglobin and erythrocyte content. The addition of blood by transfusion will be a great assistance in hastening the recovery process, as improved oxygen supply will benefit every actively functioning tissue in the body.

### SALINE TRANSFUSION

Ordinary physiological saline, containing 0.85 per cent sodium chloride, may be used, but a balanced salt solution containing the chlorides of sodium, potassium and calcium is much more desirable. This combination is known as Ringer's solution. Sodium bicarbonate added to the above salt solution will aid in controlling any acidosis such as tends to develop in severe hemorrhage and shock. These salt ions are essential to the contraction of heart muscle and other muscular tissue, exert osmotic effects, and assist the functioning of the nervous system.

A saline solution should duplicate as nearly as possible the composition of blood plasma. Drug companies supply tablets containing the ingredients of Ringer's mixture necessary for preparing any desired amount of solution on brief notice. The solution should be warmed to body temperature before injection and, needless to say, must be free of any particles of insoluble material. Injection can be accomplished by means of pressure or gravity. Gravity injection is most desirable, as it eliminates to a large extent too rapid a flow and provides a more steady, better regulated flow.

### MANIPULATION OF BLOOD IN TRANSFUSION

Blood properly selected and transfused into the circulation of another animal of the same species will circulate in its new environment without suffering injury or inflicting injury and will contribute its functional activity to the recipient animal body for a period of weeks or several months. Proper selection is made upon the basis of the compatibility of the donor blood with that of the recipient body. There must be a freedom from any tendency for the cells of either blood to become agglutinated or hemolyzed, the donor blood in particular tending to be more strongly affected, due to its small volume being balanced against the entire blood volume of the recipient. Agglutination of cells will cause circulatory obstruction by thrombosis. Hemolysis will destroy the erythrocytes and defeat, in part or entirely, the object of the transfusion procedure.

Among our domestic animals the horse, the ox and the dog are of sufficient economic or sentimental value to justify the use of transfusion of blood in disease conditions. The cat has been given very little attention as regards compatibility of blood or transfusion procedure, but the indications exist in connection with many feline diseases and study should be devoted to the question. The blood of the horse and of purebred dog donors should be examined for type or hemoagglutination properties. Mongrel dog blood appears to be free of type characteristics. Ox blood is also free of any type characteristics that may interfere with transfusion.

### BLOOD-TYPING TECHNIC

Typing of blood is a simple procedure that can be carried out successfully with the equipment available in the office of any practitioner. The procedure, outlined as follows, has proved to be the most practical and reliable:

1. Draw a 10-cc sample of blood from the recipient and also the same amount from two or more donors, or such donors as may be available. Collect blood in test-tubes and set aside to clot.

2. Decant the serum from each test-tube into small test-tubes numbered according to sample.

3. Add 5 cc of 0.85 per cent sodium chloride to the blood clot in each of the collection tubes and shake until the salt solution is bright red with suspended erythrocytes. Decant this cell suspension into tubes numbered according to the sample.

4. Place a medicine-dropper in each of the serum and cell-suspension tubes.

5. On a piece of clear window glass, place two drops of the recipient's serum at intervals along one side, the numbers of these serum deposits corresponding to the numbers of cell-suspension tubes.

6. In the first serum deposit, place a drop of the recipient's cell suspension, using the remaining serum deposits for the cells of the prospective donors. In the first deposit, the recipient's serum and cells are combined and, consequently, should always be negative, thus serving as a control in reading the remaining combinations.

7. Mix the cells and serum thoroughly in each combination by stirring with toothpicks or match-sticks, using a fresh pick or match for each combination.

8. Tilt the glass plate back and forth for a period of five to ten minutes to prevent sedimentation of the blood-cells. Ten minutes is the maximum interval required for observation.

A negative reaction is indicated by the serum-cell combination maintaining a homogenous appearance. The erythrocytes of horse blood tend to assume rouleaux or the pile-of-pennies formation that often gives the combination a faintly granular appearance that may be confused with a weak agglutination and will be observed to occur in the control combination.

A positive reaction is marked by the pronounced clumping of blood-cells, giving the serum-cell mixture a coarsely granular appearance; or, if the reaction is very pronounced, the cells may all aggregate into a single central mass which resists separation when the glass is jarred by slight blows on the edge by the palm of the hand. The clear serum will be observed surrounding the clump or clumps of agglutinated cells.

Such a positive reaction of the donor's cells in the recipient's serum contraindicates the use of such blood for transfusion. It must be remembered that blood-cells and capillary blood-vessels are of microscopic dimensions, so that macroscopic clumping of blood-cells indicates that thrombosis will occur in the first capillary area entered. The pulmonary capillaries constitute the initial point of obstruction to the passage of quickly agglutinated blood-cells administered intravenously, and vital nerve centers, such as the center for respiratory control in the medulla, will be protected to a considerable degree.

The serums of the donors may be typed against the recipient's cells in order to assure complete compatibility. This, however, is less important than the determination of a complete absence of agglutination of donor's cells by recipient's serum. The donor's serum may be strongly agglutinative for the cells of the recipient, but cause no disturbance unless a massive transfusion is given. This is explainable in terms of dilution effects, reducing the agglutinative power of the serum, as a relatively small amount of donor's serum is mixed with a large amount of recipient's blood. The reverse relation exists regarding the donor's cells, and any tendency for agglutination of these cells by the recipient's serum remains unmodified by the slight dilution effected in the course of the transfusion.

### TRANSFUSION TECHNIC

Three methods of blood transfusion are available for use: the direct, the semi-direct and the indirect. Use of the direct method requires the surgical anastomosis of an artery of the donor with a vein of the recipient, thus rendering possible the transfer of blood without exposure to atmosphere or foreign surfaces of any kind. This procedure, however, involves serious damage and destruction of the vessels used. On account of this undesirable feature, it is rarely utilized.

### SEMI-DIRECT TRANSFUSION

This procedure requires that the donor's and the recipient's circulations be brought into communication by means of tubing. A syringe is introduced to provide motive

power for a flow of blood from one circulation to the other. A great variety of semi-direct transfusion equipment is available for use in human medicine, some of which, by slight modification, is adapted to the needs of the veterinarian. We have used the Scannell syringe for transfusion in the horse and it appears to fulfill all of the requirements for such work. This syringe, as offered by the MacGregor Instrument Company, consists of a 25-cc syringe to which is attached a three-way valve that makes possible the establishment of communication between the syringe and donor, syringe and recipient, or syringe and saline solution container, or the exterior, if desired.

This type of valve enables the operator to flush the barrel of the syringe if there is a tendency for the plunger to adhere to the walls. It also affords an opportunity for discarding a syringe full of blood that may be undesirable for injection, due to the presence of air bubbles or other foreign material. The caliber of the valve openings, syringe nipples and the tubing connections is of such diameter as to offer a minimum amount of resistance to the passage of blood, thus reducing the extent of injury to the cellular elements and, consequently, the tendency to coagulate. The syringe is provided with a large finger-ring which enables the operator to support the syringe easily and safely in one hand while operating the piston of the syringe with the other.

Semi-direct transfusion in the horse presents no difficulties, as the necessary restraint, particularly of the donor, requires little effort. In cattle, trouble may often be experienced in securing the desired degree of immobilization of the donor, as the ox is more resistant to restraint and venous puncture than the horse. In our use of the Scannell apparatus, we attached three feet of tubing to the donor and recipient nipples of the three-way valve, thus allowing a range of six feet for the maneuvering of the animals. Cattle secured in stanchions provide the most satisfactory conditions for restraint. They do not require more than the above mentioned six feet of tubing connection. The large, easily accessible, jugular veins and the slow coagulation time normal to these genera are factors that contribute to the feasibility of semi-direct transfusion in bovine and equine practice. In the dog just the opposite conditions prevail and, thus, render semi-direct transfusion very difficult.

## INDIRECT TRANSFUSION

*The citrate method:* Sodium citrate is a very satisfactory anticoagulant, as it removes the lime salts of the blood by the formation of a soluble but non-ionizable salt. Oxalates are also anticoagulants, but remove the calcium by precipitation. A precipitate should never be introduced into the blood-stream. A concentration of 0.3 per cent of citrate will prevent coagulation. Citration of blood may be accomplished as follows:

1. In a container of known capacity (preferably a milk bottle) place a quantity of 3 per cent sodium citrate solution equivalent to one-tenth of the total capacity of the container.
2. Complete filling of the container with blood drawn as rapidly as possible from the jugular vein. Move the container during the course of the filling to insure a good mixture of the blood and citrate solution. With completion of the filling, the container is filled with a volume of blood and citrate solution, the concentration of sodium citrate now being 0.3 per cent.
3. Place the container of citrated blood in warm water to maintain a temperature equivalent to that of the body, or slightly above, until ready for injection into the recipient's vein.

We have found the following equipment very practical and satisfactory for indirect transfusion work in the horse and ox:

A milk bottle constitutes our container, it being closed with a No. 26 two-hole rubber stopper. One hole of this stopper is fitted with a ten-inch length of monel metal tubing having an outside diameter of 7/25 inch. The second hole is fitted with a 2½-inch length of this tubing. Rubber tubing (3½ feet of 6/25 inch-1/25 inch side wall) attached to the outlet of the short tubing

adapts the bottle for gravity feed. When attached to the long metal tube and with a pressure bulb connected to the short tube, the bottle may be used for pressure injection.

The advantages of this arrangement consist of (1) the ready availability of a container under all conditions and (2) the moderate first cost of the stopper, tubing and pressure bulb. The standard size of mouth openings in all milk bottles enables a variety of different capacity containers to be used with the same injection stopper.

A 6-gauge bleeding needle proves very satisfactory for the collection of blood from horse or ox, for citration or defibrination. Although it is a needle of rather formidable dimensions, it will be found that its introduction into the jugular vein of the mature horse or ox is easily accomplished. It may be necessary to nick the skin, but usually it can be thrust directly through the skin into the well-distended vein. The rapid flow of blood favors good citration and counteracts the tendency for coagulation in the needle. In removing the needle, subcutaneous escape of blood may be prevented by "pushing the animal off on the needle" rather than drawing the needle out. The compression of the skin against the vein wall will prevent the gush of blood that might fill the pocket of skin when the needle is withdrawn without support of the hand against the skin surrounding the needle.

For introducing the blood into the equine or bovine recipient, a 12-gauge needle has proved to be most satisfactory. Both the bleeding and transfusing needles should be about three inches in length, as the long needle may be placed in the vein deeply enough to insure against the danger of dislodgment.

*Preparation of defibrinated blood:* Defibrination is most conveniently accomplished by shaking, the container being filled to about two-thirds capacity to allow sufficient movement. Horse blood requires about twelve minutes for the completion of coagulation reaction; ox blood approximately seven minutes. It therefore requires an interval of ten minutes for ox blood and 15 minutes for horse blood for the complete shaking out of all fibrin. The agitation of blood during the course of the coagulation reaction serves to check any clotting action, as the fibrin is disorganized and becomes aggregated into masses that float on the surface of the blood. The fibrin is removed by straining the blood through a double layer of cheesecloth. The straining operation is the most objectionable feature associated with the use of defibrinated blood, as it subjects the blood to rather extensive handling, which predisposes to hemolysis and contamination. In contrast, the citrate method involves a minimum amount of manipulation, as the blood may be drawn directly into the transfusion bottle through the tubing connection, which can be connected immediately with the needle in the vein of the recipient following the collection, if so desired.

### DISCUSSION

DR. J. D. RAY: About what percentage of horses are unsuitable as donors?

DR. AMADON: I am unable to state the percentage, but I can state that the danger probably is limited, as the number of horses which show strong type reactions is not great. If you are working with valuable animals, I would certainly advise testing the blood to insure against selecting an animal that will agglutinate all cells and consequently be a very unsuitable recipient. Such an animal, when transfused, unquestionably dies as a result of the transfusion. I will say, though, the percentage is not high. You will not find strongly reacting blood in the horse. Such animals do exist, however.

DR. W. J. WILLIAMS: How does normal serum compare with normal saline?

DR. AMADON: Normal serum is preferable to saline because it contains 6 to 7 per cent of protein material, which will contribute to the retention of the serum within the circulation of the recipient. If you have serum available, I would certainly advise it in preference to saline for transfusion purposes; that is, if you are attempting to restore volume.

DR. RAY: All so-called normal horse serum that is sold commercially must be heated to a certain temperature to eliminate the possibility of virus infections. What is the effect of that?

DR. AMADON: If the serum is heated to a temperature that results in the precipitation of the proteins, of course, this practice would tend to reduce the protein content, but I would not say that it would reduce the value of the serum to any great extent.

DR. RAY: The serum is not heated to the point of precipitation, but only to 58 to 59° C., just below the temperature that would render the serum toxic.

DR. AMADON: Then there is no loss of protein value. It would still have the osmotic value which is the important feature of serum.

# Some Observations on the Nature and Transmission of Enzootic Bronchopneumonia (Pneumoenteritis) of Dairy Calves*

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Enzoötic bronchopneumonia, septic pneumonia, or pneumoenteritis of young animals has been encountered by workers since the middle of the last century. The disease is reported to be common among dairy calves less than six months of age and losses also have been recorded in lambs, pigs, kids and foals. Enzoötic bronchopneumonia has been described by Hutyra and Marek[1] in European countries, and by Edmonds[2] in the British Isles, South Africa and other places throughout the world. Roberts[3] gave a recent account of tropical pneumoenteritis of calves in the Dominican Republic and in the states of Sao Paulo and Minas Geraes, Brazil.

This writer reported the disease common in calves from a few days to six months of age and states that, regardless of the treatment used, 80 per cent of the clinical cases and all of the serious cases succumb to the ailment. Various writers, including Poels, Jensen, Van den Maegdenberth and Lienaux (quoted by Hutyra and Marek[1]) concluded that a bacillus resembling the swine plague organism often was responsible for a disease which they described as enzoötic or septic pneumonia of calves. Other microörganisms considered to be the cause of enzoötic bronchopneumonia of calves include species of *Streptococcus* and *Staphylococcus, Bacillus pyogenes, Bacillus pyocyaneus,* and bacteria of the colon-typhoid group.

Opportunity has been afforded for making observations on the nature of an enzoötic pneumonia occurring in calves and yearlings on dairy farms in Florida. This condition corresponds in clinical symptoms, postmortem lesions, bacteriological findings and occurence to enzoötic, septic bronchopneumonia or pneumoenteritis as described by Poels and others.[1] The disease has many features in common with that occurring in South America and in the Dominican Republic. Enzoötic bronchopneumonia of calves as observed in Florida may assume either an acute or chronic form. Both types may be present simultaneously within the same herd.

The disease may occur at any time throughout the year, but is more frequent during the warm, moist season of the summer months. It abates to a large extent during the dry periods of the year. Chronic cases which have their incipient stages during the wet periods of the year often linger for weeks without regard to season or moisture conditions. In the herds under observation there is a tendency for the condition to recur year after year, causing heavy losses among the young calves. The mortality rate frequently amounts to 60 or 70 per cent of the annual calf crop. Further loss is sustained since animals that survive the infection are underdeveloped, unthrifty and unprofitable.

When first introduced into a herd, the disease usually starts among the young calves as an acute infection of the gastrointestinal tract. During the incipient stages, infected calves show a roughened coat and refuse to eat normally. They exhibit an elevation of body temperature, diarrhea and weakness. Symptoms of gastroenteritis may become intense, in which instance many cases develop symptoms of septicemia and die three to seven days later. If the calves survive for a few days, symptoms of pneumonia usually develop. The calves cough, exhibit a mucopurulent nasal discharge, swelling of the sublingual region, labored respirations and emaciation. A persistent fetid diarrhea contain-

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ing blood coagula is characteristic of the early stages.

Periods of temporary improvement may be followed by a prolonged illness of several weeks duration characteristic of the chronic form. During the chronic stages, the acute symptoms of gastroenteritis disappear and the appetite and general appearance may improve. As the chronic condition progresses, the animals continue to cough, and are easily exhausted. They show evidence of nutritional disturbance and anemia, regardless of the improved appetite. Infected animals remain lying a great portion of the time. They assume a characteristic position of sternal recumbency, with head and neck extended and resting the cervical and mandibular regions on the ground.

The infected animals may linger for weeks, exhibiting symptoms of bronchopneumonia. In the more advanced cases oral breathing is common, the tongue protrudes, the mouth contains a frothy exudate, and the animals show evidence of a grave inspiratory and expiratory pulmonary dyspnea. Fatal cases exhibit a tenacious bilateral nasal discharge and succumb with pronounced symptoms of pulmonary edema and pneumonia.

Postmortem examinations of those animals which succumb during the acute stage of the condition show lesions of a violent gastroenteritis, pulmonary congestion, and inflammation of the internal organs. The liver is enlarged, friable and yellow. Postmortem examinations of calves, made during various stages of the chronic form, show a progressive bilateral hepatization of the lung tissue. The solidifying process of the lungs begins in the lower apical lobes and gradually extends to involve the cardiac and diaphragmatic lobes.

Fatal cases show solidification of the entire lung tissue with the exception of a very small portion of the dorsal border of the diaphragmatic lobes. The intralobular connective tissue of the lungs is thickened and yellowish in appearance and areas of necrosis appear throughout the lung substance. There may be present an extensive adhesive pleurisy with a fibrinous exudate in the pleural cavity. The trachea and bronchi are filled with a frothy exudate.

Due to the economic loss resulting from year to year in the calf crop of infected herds, studies were undertaken regarding the nature and transmission of the disease as it occurred under local conditions. Various bacteria were found associated with the diseased tissues. *Escherichia coli* was isolated from the intestinal tract and from the blood-flecked droppings of acute cases. *Pasteurella boviseptica* was the predominating bacterial species encountered upon microscopic and cultural examination of diseased lung tissue. The virulency of this organism for small laboratory animals was typical of the *Pasteurella* group. Species of *Staphylococcus* and *Penicillium* were isolated from the lung tissue of several calves that were autopsied in advanced stages of the chronic form of pneumonia.

It has been considered that a primary etiological relationship exists between species of *Pasteurella* and enzoötic bronchopneumonia of young animals. Since *P. boviseptica* was readily isolated from diseased lung tissue of calves affected with the chronic form of enzoötic bronchopneumonia, it was deemed important to study the relation of this organism to the infection.

Strains of the organism used in studying the relation between *P. boviseptica* and enzoötic bronchopneumonia were secured by culturing diseased lung tissue of calves that died during chronic stages of the infection. Other strains were secured by aspirating secretions from the nasopharyngeal region of calves in various stages of pneumonia. *P. boviseptica* was isolated from these secretions by inoculating laboratory animals and by plate cultures. Other strains were obtained by culturing the heart-blood of laboratory animals which received injections of edematous tracheal exudate and minced portions of affected lung tissue of calves that died of enzoötic bronchopneumonia.

After isolation, the various strains of *P. boviseptica* were grown and maintained on ferric salts agar, meat infusion broth containing ferric ammonium citrate, Kracke

and Teasley's blood culture media, Difco stock culture agar containing blood and in whole blood. The hearts and spleens of rabbits that succumbed after exposure to *P. boviseptica* were ligated and removed aseptically. These tissues were used to maintain certain strains of the organism. Stock cultures maintained in the laboratory were transferred every fortnight and either stored at room temperature or under refrigeration. *P. boviseptica* grown and maintained under these conditions was highly virulent for small laboratory animals as proven by intraperitoneal or intravenous inoculations using minute quantities of the stock cultures.

Attempts were made to reproduce cases of enzoötic bronchopneumonia by exposing calves to *Pasteurella* that were isolated from diseased lung tissue. Young calves, secured from herds where pneumonia did not occur, were exposed to the organism by way of the digestive tract. Amounts varying up to 200 cc of a 24-hour bouillon culture were given as a drench. Calves also were drenched with minced internal organs of laboratory animals that succumbed to injections of *P. boviseptica*. The diseased lung tissue of calves that died in advanced stages of enzoötic bronchopneumonia were minced in saline solution and administered to young healthy calves as a drench. No evidence of pneumonia developed in any of the calves thus exposed. No difference was found as to the type or character of the media used in growing or maintaining *Pasteurella*, as influencing the development of pneumonia in young healthy calves exposed to the organism. Results of this test led to the belief that *P. boviseptica* was not the primary etiological agent of enzoötic bronchopneumonia of calves.

Exposures of calves by contact with active cases of enzoötic bronchopneumonia were made. A total of eight calves in various stages of the condition were selected from naturally occurring field cases and confined with a similar number of young healthy calves. The exposure was made in specially constructed isolation pens of concrete[4] which were cleaned each day. Acute gastroenteritis typical of early stages of the naturally occurring disease developed in these test calves several days following exposure. *E. coli* was isolated from the diarrheal discharge of the test calves.

The recovered organism produced acute gastroenteritis in young healthy calves when bouillon cultures were given as a drench. Although diarrhea typical of early stages of the disease developed in the calves thus exposed, typical enzoötic bronchopneumonia, as observed under field conditions, did not develop in the limited number of calves exposed. Nasal and bronchial secretions of animals suffering from enzoötic pneumonia, minced portions of affected lung tissue and bacteria-free filtrates of these materials did not reproduce the disease when healthy calves were exposed thereto by the nasal and oral routes.

It was concluded that *E. coli* was instrumental in producing symptoms characteristic of the early stages of enzoötic broncho-pneumonia. It appeared that predisposing factors not present in the concrete isolation pens, but incident to environment of infected herds, were necessary for successful transmision of the typical chronic form of the disease.

Examinations of clinical cases of pneumonia showed that many of the animals were infested with the blood-sucking louse, *Linognathus vituli*. Microscopic examinations of fecal samples from calves of infected herds showed the presence of coccidia (*Eimeria* sp.) and nematode ova. Fecal examinations of older calves on affected premises revealed a heavy parasitic infestation. Postmortem examinations of yearlings which survived an acute attack showed that the herd was infested with several species of internal parasites, including hookworm, *Bunostomum phlebotomum;* tapeworm, *Moniezia benedeni;* nodular worm, *Oesophagostomum radiatum;* stomach worm, *Haemonchus contortus;* whipworm, *Trichuris ovis;* lungworm, *Dictyocaulus viviparus;* and the filaria, *Setaria labiato-papillosa.*

An attempt was made to reproduce bronchopneumonia by employing insanitary methods of rearing calves similar to those

observed under field conditions. Young healthy calves were secured from herds not infected with the disease. They were confined and reared on a small, shaded lot heavily contaminated with droppings of animals showing clinical manifestations of the acute and chronic types of bronchopneumonia. Typical bronchopneumonia, similar to those cases observed under natural conditions, subsequently developed in a number of the test calves. Postmortem examinations of such reproduced cases showed lesions similar to those occurring in the field.

Results of the test showed that predisposing factors incident to crowded, insanitary, permanent calf-lots were necessary to reproduce typical cases of bronchopneumonia in calves.

Field and experimental observations indicate that enzoötic bronchopneumonia of dairy calves depends upon a number of predisposing factors. This condition appears where calves are confined in crowded, insanitary, permanent lots and is more prevalent during the warm, moist seasons of the year. These conditions are favorable to development of various bacterial infections of the gastrointestinal tract and the umbilicus, and to infestations with external and internal parasites. Calves kept under such conditions frequently are undernourished. These weakening influences lower the body resistance sufficiently to permit development of pneumonia.

No single predisposing factor capable of reproducing the typical pneumonia has been found in a limited number of exposed calves. Independent infections with *P. boviseptica, E. coli, Eimeria* sp., lungworm, or other single factors have not reproduced the typical bronchopneumonia under experimental conditions. Further work will be necessary before definite statements can be made regarding the rôle of each of these, and other predisposing factors, in producing bronchopneumonia. The disease has not been observed on premises where strict sanitary methods of rearing calves are practiced. The incidence of enzoötic bronchopneumonia in infected herds has been reduced in direct proportion to hygienic methods employed in rearing calves.

## SUMMARY

1. Enzoötic bronchopneumonia causes heavy loss among dairy calves confined in crowded, insanitary, permanent lots.

2. *Escherichia coli, Pasteurella boviseptica,* and species of *Staphylococcus* and *Penicillium* were found to be associated with the condition.

3. Older calves affected with the chronic form of enzoötic bronchopneumonia were found to be infested with various species of internal parasites, including: coccidia, *Eimeria* sp; hookworm, *Bunostomum phlebotomum;* whipworm, *Trichuris ovis;* tapeworm, *Moniezia benedeni;* nodular worm, *Oesophagostomum radiatum;* lungworm, *Dictyocaulus viviparus;* stomach worm, *Haemonchus contortus;* filaria, *Setaria labiato-papillosa;* and the external blood sucking louse, *Linognathus vituli.*

4. No single predisposing factor has been found that reproduced typical bronchopneumonia as observed under field conditions.

## ACKNOWLEDGMENT

The author is indebted to Dr. G. Dikmans, of the Bureau of Animal Industry, for identifying several species of internal parasites encountered.

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## DISCUSSION

DR. G. A. ROBERTS: I am the Roberts that the speaker spoke of as having observed pneumoenteritis in Brazil, Puerto Rico, the Virgin Islands and, more recently, in the Dominican Republic. I am not sure whether we are dealing with the same thing that Dr. Sanders is or not, but we have a tremendous amount of so-called pneumoenteritis.

We have some peculiar conditions that I do not believe exist in this country. In the first place, in both Brazil and in the Dominican Republic, we find this trouble only in purebred

and grade calves. We do not see it at all in native calves. Furthermore, we do not see it in calves that are running loose in pasture. In those countries, the custom is to milk only once a day, and the calves are therefore brought up in the evening to be held over until milking time early in the morning. They are crowded together either in pens or in sheds. It is in these calves that this trouble occurs, and in some cases, as was reported, as many as 80 or 90 per cent of the herd will become involved.

Our belief is that the disease is not an entity at all—that it is a combination of various factors, and there does not seem to be any one of greater predominance than the other. There are two or three in this case, so we have been able to get nowhere in trying to identify the disease as a specific entity in itself.

We have observed, too, that the cases may be purely pneumonic in character, they may be purely enteric in character, or, as in most cases, there may be a combination of the two —pneumonia and enteritis—and therefore we think the term pneumoenteritis is very appropriate.

In measures of prevention and cure, we have observed wonderfully good results in our cases where we can induce the owner of these animals not to keep the calves in. But because of the fact that it is so difficult for them to do so, we are unable to do very much in the prevention of unhygienic measures by allowing them to run out in pasture. Those who have turned their calves back out into the pasture do so as soon as they have nursed, because they say that the cows will not give milk if they do not let the calves nurse first. Therefore, the difficulty comes in getting them to do as we would do in this country.

We have attempted a number of things, and at the present time we have about five or six different groups of calves in which we are using various biological products. The principal one that we are depending upon is the bacterial aggressin of hemorrhagic septicemia. Our own opinion is that very probably there is a lowered vitality from the infection with the anaplasmosis organism, and the hemorrhagic septicemia organisms that, in themselves, are not serious, lower the vitality sufficiently for other organisms to come in and cause trouble.

Just one word, then, as to our observations in trying to treat these cases. We have observed some beneficial results from what is a popular remedy down there, in the use of large doses of bicarbonate of soda, in an attempt to make the conditions as unfavorable as possible for the hemorrhagic septicemia organism to grow. We have likewise used, with apparently good results, in some cases, sulfanilamide, as it is used in this country.

## Sodium Bicarbonate Mine Found

The finding of a natural deposit of sodium bicarbonate far under an ancient California lake-bed has been reported by the Smithsonian Institution.

## Meat from 70 Million Animals Gets Federal Inspection Annually

Fifty years ago, there was no federal meat inspection. Today, the United States Department of Agriculture inspects about 70,000,000 meat animals annually. Immediately before and at the time of slaughter, each of these food animals receives a thorough inspection by one or more trained veterinarians—graduates of accredited colleges.

The Bureau of Animal Industry, in announcing a new film strip on meat inspection, recalls that the primary necessity for federal meat inspection came from European governments, which looked with disfavor on United States meat that carried no official evidence that it had come from healthy animals.

In 1890, Congress established a meat-inspection service. Shortly afterward, it was broadened to apply to all slaughtering establishments that prepared meat for interstate and also foreign trade.

The present meat-inspection law was enacted in 1906. This law still further extended the federal meat inspection to include all meats and meat food products of establishments that ship their products in interstate or export trade. However, the law exempts farmers from federal inspection, and to some extent local butchers and meat dealers who ship their products across state lines in serving their own customers. Any meat business entirely within the boundaries of a state is exempt. It works out that about two-thirds of the country's meat supply receives federal inspection.

There are seven essential parts to federal meat inspection. These are: sanitation of the establishment, inspection before slaughter, postmortem inspection, products inspection, laboratory inspection, disposal of condemned material, and labeling. Although most live stock offered for slaughter is healthy, veterinarians in the meat-inspection service must be on the watch constantly for about 50 diseases or abnormal conditions that prevent animals or their carcasses from receiving a clean bill of health.

# Chemical Studies of the Blood of Normal Cows*

*By* O. F. REIHART,† *Omaha, Neb.*

To secure a basis for biochemical diagnostic procedures as applied to domestic animals, the following investigation was undertaken on the normal cow. It was deemed advisable to secure quantitative data with regard to various blood constituents in lactating and non-lactating cows, both pregnant and heifers. Furthermore, to determine the effect of the act of milking, the blood changes before and after milking have been studied.

Although similar values have been published by previous investigators, a reëxamination seemed desirable anyway, with the aid of more refined and widely accepted analytical procedures.

The animals employed in this investigation all belonged to the same herd of registered pedigreed Holstein-Friesian cows, which was free from tuberculosis, Bang's disease and mastitis, and was generally in good condition. The animals were grouped according to age to ascertain the influence of this factor.

The heifers were fed a mixture consisting of:

| | |
|---|---|
| Corn | 1,600 lbs. |
| Oats | 1,400 lbs. |
| Soybeans | 100 lbs. |
| Salt | 30 lbs. |
| Bone meal | 30 lbs. |
| Limestone | 30 lbs. |
| Linseed | 100 lbs. |

The lactating and the pregnant cows were fed a mixture consisting of:

| | |
|---|---|
| Corn | 1,200 lbs. |
| Oats | 1,200 lbs. |
| Bran | 1,000 lbs. |
| Cottonseed meal | 200 lbs. |
| Soybean oil meal | 200 lbs. |
| Linseed oil meal | 300 lbs. |
| Steam bone meal | 40 lbs. |
| Salt | 40 lbs. |
| Limestone | 40 lbs. |

The blood for analysis was drawn from the jugular vein. Two 25-cc samples were taken: one into a vial containing as anticoagulant four drops of a 20 per cent citrate solution; the other sample was collected in a clean vial and allowed to clot overnight. The citrated blood was used for determining sugar, non-protein nitrogen, urea nitrogen, uric acid, preformed creatinine, cholesterol and chlorides. There serum from the second sample was used for the determinations of sodium, potassium, calcium, magnesium, and inorganic phosphorus.

The blood sugar,[1] non-protein nitrogen, uric acid and creatinine were determined by the Folin method[2]; urea nitrogen by the Haskins procedure[3]; cholesterol by the Liebermann-Burchard[4]; color reaction; sodium by the procedure of Weinbach[5]; potassium by the method of Kramer and Tisdall[6]; calcium by the method of Wang[7]; magnesium by the method of Kallinikowa[8]; and phosphorus by a modification of the Doisy-Bell procedure[9]; the chlorides were determined on the whole-blood filtrate by the procedure of Whitehorn.[10]

## RESULTS

The analyses were made on 60 lactating and 20 non-lactating cows. The full data of these analyses would occupy too much space; in table I we present the average results for each group. No definite deviations were found in the blood sugar under the conditions of this study, but heifers tend to have a higher sugar value. The non-protein nitrogen also shows no marked variation except perhaps for a slight tendency to be higher in pregnant cows. Neither has there been any marked change in the blood urea nitrogen, but the non-lactating cows have a somewhat higher blood urea value.

As far as uric acid is concerned, the pregnant cows seem to have a lower value, while heifers a definitely higher value than the lactating cows. No variation was found in the blood creatinine. The cholesterol content of the blood is definitely greater in the lactating than in the non-lactating cows, and seems to be affected by milking. It is not only raised somewhat during the process of milking but even two hours later it shows a still greater increase over the pre-milking period (two hours before).

As far as the inorganic constituents of the blood are concerned, there are prac-

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†With the technical assistance of Anna Thurston, A. B., Omaha, Neb.

TABLE I—*Average values for various blood constituents.*

| | Blood Analyses | Condition of Cows | | | | |
|---|---|---|---|---|---|---|
| | | Lactating (60) | | | Non-Lactating (20) | |
| | | 2 Hours Before Milking | At Milking Time | 2 Hours After Milking | Pregnant Cows | Heifers |
| Mgm % Whole Blood | Sugar | 81.3± 3.2 | 79.2± 4.3 | 82.7± 3.7 | 81.9± 3.9 | 90.2± 5.8 |
| | N P N | 24.4± 2.7 | 24.7± 4.5 | 21.0± 4.0 | 30.9± 3.5 | 25.9± 2.5 |
| | Urea N | 5.5± 0.7 | 5.7± 0.9 | 5.9± 0.8 | 6.5± 1.4 | 6.6± 1.0 |
| | Uric Acid | 2.8± 0.3 | 3.3± 0.7 | 3.1± 0.8 | 2.3± 0.5 | 4.1± 0.9 |
| | Creatinine | 1.4± 0.09 | 1.4± 0.07 | 1.4± 0.2 | 1.5± 0.1 | 1.4± 0.02 |
| | Cholesterol | 183 ±29.2 | 194 ±26.4 | 209 ±28.0 | 161 ±24 | 156 ±18.9 |
| | Cl | 306 ±17 | 294 ±31.8 | 246 ±15.0 | 285 ±25 | 331 ± 6.0 |
| Mgm % Serum | Ca | 11.3± 0.4 | 11.0± 0.9 | 11.6± 0.9 | 11.4± 0.6 | 11.8± 0.6 |
| | Mg | 1.8± 0.4 | 1.8± 0.3 | 2.0± 0.4 | 1.7± 0.2 | 2.2± 0.3 |
| | Na | 351 ±15.8 | 355 ±14.9 | 380 ±10.2 | 380 ±16 | 371 ±10.2 |
| | K | 25.2± 1.4 | 24.0± 1.5 | 24.6± 1.9 | 24.7± 2.5 | 24.1± 1.5 |
| | P | 5.6± 0.7 | 5.4± 0.6 | 5.5± 0.6 | 5.2± 0.6 | 4.8± 0.4 |

tically no changes in the calcium, magnesium, potassium, or phosphorus. The sodium in the lactating cows is somewhat lower but is definitely increased following milking, while the chlorides show a decrease of about 2υ per cent under this condition.

A study of the ratios of the inorganic constituents of serum throws some interesting light (table II). Thus, the calcium/phosphorus ratio, which is relatively constant in the lactating cows, not being appreciably affected by milking, is decidedly lower than in heifers, because of their higher phosphorus content. It is interesting to observe that in pregnant cows this ratio is much lower than in heifers and approaches more nearly that of the lactating cow, due to a gradual rise in the blood inorganic phosphorus.

The calcium/magnesium ratio shows a certain shift after milking, the decrease in the ratio being due to an increase in magnesium. Finally, the sodium/potassium ratio shows that, two hours after milking, these elements are present in the cow's blood in the same relative amounts as in the non-lactating cows, but before milking the sodium decreases. It increases during the milking process and continues to do so afterwards, because two hours after the milking the ratio is reëstablished to the level prevailing in the non-lactating animal. These studies indicate a significant shifting of ions in the organism apparently associated with the production of milk.

TABLE II—*Ratios of inorganic constituents of serum.*

| Condition of Cows | | Ratios | | |
|---|---|---|---|---|
| | | Ca:P | Ca:Mg | Na:K |
| Lactating | 2 hours before milking | 2.02 | 6.38 | 13.8 |
| | At milking time | 2.04 | 6.08 | 14.8 |
| | 2 hours after milking | 2.07 | 5.71 | 15.4 |
| Non-Lactating | Heifers | 2.46 | 5.41 | 15.4 |
| | Calvy cows | 2.15 | 6.74 | 15.5 |

## SUMMARY

Blood of non-lactating and lactating cows has been analyzed for sugar, non-protein nitrogen, urea nitrogen, uric acid, preformed creatinine, cholesterol, sodium, potassium, calcium, magnesium, chloride and phosphorus. Blood from the lactating cows was taken two hours before and two hours after milking and at the time of milking. Milking causes the cholesterol to rise and the chloride to fall. This tendency manifested itself at the time of milking and continued two hours later.

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# Effect of Prontosil and Sulfanilamide on Brucella Abortus Infection in Two Cows*

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Since sulfanilamide, prontosil and similar chemical compounds have been used extensively in the treatment of bacterial (especially streptococcic) infections in both human and veterinary medicine, it was thought that these compounds might have favorable results in the treatment of Brucella infections in animals. The results of this experiment are presented as a preliminary report, as the data obtained are by no means conclusive.† It will be necessary to make a much more comprehensive study of these and similar compounds before definite conclusions can be drawn.

### CASE 1

This was a purebred Jersey cow, three years old, obtained from a heavily infected herd. The animal became a reactor to the agglutination test during her first pregnancy and aborted a seven-month fetus. At the time of purchase, March 8, 1938, the animal had been pregnant again for about two months and was in good physical condition. The agglutination test was positive in a 1:400 dilution, and *Brucella abortus* was being eliminated in the milk from all quarters. The blood and milk agglutination reactions, phagocytic index,[1] and cultural findings of the milk of this animal, while in our possession, are given in table I.

Treatment of this animal with a 5 per cent prontosil solution was begun on March 21, 1938. Previous to this time, temperatures were taken for several days in order to determine the normal temperature range. The temperature was found to vary from 100.8° to 102.0° F. At 4 p. m., on March 21, 100 cc of prontosil was injected subcutaneously and intramuscularly. The temperature began to rise about two hours after administration and reached its highest point (104° F.) at 9 p. m., after which it gradually subsided. On March 24, another 100 cc of prontosil was injected in the same manner. This time the temperature did not rise above 103.4° F. Injections of 125 cc of prontosil were given on the following dates: March 25, 26, 27 and 28; April 16, 20, 21, 24 and 28; May 5 and 10. No appreciable rise in temperature was noted following any of these injections except on April 16, when a temperature of 104.0° F. was recorded.

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†This study was supported by a grant from the Horace H. and Mary A. Rackham Fund.

TABLE I—*Laboratory findings, cow 1.*

| DATE | BLOOD AGGLUTINATION | MILK AGGLUTINATION* | | | | PHAGOCYTIC INDEX† | | | | MILK CULTURES | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | R.F. | R.R. | L.F. | L.R. | 1 | 2 | 3 | 4 | R.F. | R.R. | L.F. | L.R. |
| 3- 9-38 | +++++ | +++ PT | +++ PT | ++ PT− | +++ PT | 8 | 9 | 6 | 2 | + | + | + | + |
| 3-15-38 | | ++ PTT | ++ PT− | ++ PT− | ++ PT− | | | | | + | + | + | + |
| 3-16-38 | +++++ | | | | | 0 | 0 | 4 | 21 | | | | |
| 3-25-38 | +++++ | ++ PT | ++ PT− | +++ P− | +++ PT | 15 | 8 | 2 | 0 | + | + | + | + |
| 4- 4-38 | +++++ | +++ PT | ++ PT− | ++ PPT | +++ PT | 13 | 7 | 5 | 0 | + | + | Mld | + |
| 4- 8-38 | +++++ | | | | | 11 | 8 | 6 | 0 | | | | |
| 4-16-38 | +++++ | ++ PTT | + PT−− | ++ PT− | +++ PT | 4 | 5 | 9 | 7 | + | + | + | + |
| 4-23-38 | +++ PT | | | | | 0 | 4 | 11 | 0 | | | | |
| 5-10-38 | +++++ | ++ PTT | + PT−− | ++ PT− | + PT−− | 5 | 9 | 10 | 0 | + | + | + | + |

*R. F. = right fore quarter. R. R. = right rear quarter. L. F. = left fore quarter. L. R. = left rear quarter.
†1 = neutrophiles containing more than 40 bacteria. 2 = neutrophiles containing 20 to 39 bacteria. 3 = neutrophiles containing 1 to 19 bacteria. 4 = neutrophiles containing no bacteria.
Mld = mold.

On April 13, 125 cc of prontosil was injected intravenously. This amount was injected by gravity as fast as gravity would pass it through a 14-gauge needle. The animal did not show any signs of distress and the temperature did not rise above 102.6° F. following the treatment. The animal urinated within five minutes following administration. The urine was decidedly red in color.

In addition to the prontosil, the animal was given 40 gm of sulfanilamide morning and night, from April 13 to 20, inclusive. This was administered in 1½-oz. gelatin capsules, each capsule containing 20 gm.

### CASE 2

This was a grade Jersey cow, five years old, purchased from another heavily infected herd. The animal had aborted twice, and showed the organism in all four quarters of the udder. The blood agglutination had been positive in a 1:400 dilution for about 24 months prior to purchase. The animal was in very good physical condition and about five months pregnant at the beginning of the experiment.

The treatment received by this animal consisted of several injections of 50 cc of 5 per cent prontosil into the right quarter of the udder. Injections were made on the following days: April 13, 16, 20 and 24; May 5 and 10. A rise in temperature occurred four to eight hours after each injection. The highest temperature (105.6° F.) was recorded six hours following injection on April 20. After each injection, a severe induration of this quarter which would persist for three to four days was observed. The milk from this quarter would be colored red for 36 to 48 hours after injection, the degree of discoloration subsiding gradually. The animal was slaughtered May 18, 1938. Microscopic changes in the treated quarter could not be observed. No microscopic examinations of the udder were made. Blood and milk reactions of this animal are given in table II.

### DISCUSSION

The results in both experiments indicate that the administration of prontosil, either subcutaneously, intramuscularly, or intravenously, will cause a rise in temperature, the onset and duration of this depending somewhat on the method of administration. Whether sulfanilamide given orally alone, as was done in the case of cow 1, will cause such a reaction could not be determined. The animal was injected with prontosil also during that time. It will be noted that the phagocytic index was raised after prontosil had been administered. After sulfanilamide (80 gm daily) had been given to cow 1 for a few days, the animal appeared somewhat weak, the hair became rough, and the appetite was decreased at times. It appears that we were approaching the maximum tolerance dose for an animal of this size (the cow weighed 790 pounds at time of slaughter) over a longer period of time.

It has occurred to us that, in the case of cow 2, the rise in temperature may have been due to the induration in the udder rather than to the prontosil itself, as the phagocytic index was not appreciably raised. The bacteriological findings do not indicate that the presence of the organism in the udder was reduced. It is regrettable that these same animals could not have been kept long enough to observe any eventual

TABLE II—*Laboratory findings, cow 2.*

| DATE | BLOOD AGGLUTINATION | MILK AGGLUTINATION* | | | | PHAGOCYTIC INDEX† | | | | MILK CULTURES | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | R.F. | R.R. | L.F. | L.R. | 1 | 2 | 3 | 4 | R.F. | R.R. | L.F. | L.R. |
| 4– 4–38 | +++++ | ++ P T– | ++ P T– | + P T– | + P T– | 0 | 0 | 14 | 11 | + | + | + | + |
| 4–16–38 | +++++ | + P T– | ++ P T | + P P T– | + P T– – | 5 | 7 | 9 | 4 | + | + | + | + |
| 4–23–38 | +++ P T | | | | | 4 | 19 | 2 | 0 | | | | |
| 5–10–38 | | + P T– | + P T– | P P T– – | P T– – | | | | | + | + | + | + |
| 5–17–38 | +++ P T | + P T– | + P T– | + P T– | P T T– – | 5 | 12 | 5 | 3 | + | + | + | + |

*For key to abbreviations, see table I.
†See table I.

influence on the course of the infection. It is believed that further experiments in the treatment of brucellosis in animals with these products are indicated, especially since the reports of Richardson[2] indicate favorable results in the treatment of undulant fever in man, and in view of the results obtained by Montgomerie[3] in the treatment of infected guinea pigs and goats. Maximum dosages, frequency and best methods of administration, as well as comparisons of various similar products should be studied more extensively before any definite conclusions can be arrived at.

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## More Information Needed on Fur Farming Business

The United States Department of Agriculture would like to know how many fur farms there are in the United States and how much income farmers derive from furs of captive animals. Only estimates are available, because the farm census does not take a count of fur farms. To get accurate information, more than 20,000 fur farmers have been queried.

Because fur animals were trapped without thought of conservation, this country has changed in a little more than 100 years from the world's leading fur exporter to the world's largest fur importer, producing only about one-third of the furs it uses.

To meet the demand for more furs, according to Frank G. Ashbrook, in charge of fur resources for the Bureau of Biological Survey, farmers have begun raising such animals as foxes, minks, martens, and beavers in captivity. He estimates that this year some 300,000 silver fox and 200,000 mink furs will be sold by fur farmers. They will receive about $13,000,000, or about 20 per cent of the total value of all raw furs sold this year. Most of the farms are less than ten years old.

## Cows in Well Built Barns Need No Artificial Heat

Artificial heating of dairy barns is not necessary, even in the colder parts of the United States, and is not even desirable when the cost is left out of consideration. If a dairy barn filled with cows is well built and well insulated, and if the ventilation is regulated to prevent drafts, heat from the cows will keep the stable warm enough for best production.

At temperatures of 50 to 55° F. dairy cows do better than at 60 to 65° F. Cows in heated barns may give a little more milk, but not enough to pay for the trouble and expense of keeping them warmer. Cows kept at moderate temperatures seem more comfortable, have better appetites and remain healthier than cows in stables with high temperatures. Any illness of dairy cows cuts production. Excessive drafts are likely to cause pneumonia.

These are some of the high lights of an investigation of the relation of stable environment to milk production, in which M. A. R. Kelley, of the federal Bureau of Agricultural Engineering, coöperated with I. W. Rupel, of the University of Wisconsin, in practical comparison of matched herds of Wisconsin dairy cows in stables where temperatures could be controlled carefully. The tests covered two seasons.

## Retired

Drs. Duncan R. Gillies (Ont. '91), of Washington, D. C.; Alexander G. Johnstone (Chi. '15), of Chicago, Ill.; Michael J. Murphy (Amer. '96), of New York, N. Y., and Bernhard P. Wende (Ont. '92), of Buffalo, N. Y., were retired from the service of the U. S. Bureau of Animal Industry on September 30, 1938.

During October and November, the following veterinarians were retired from the service: Drs. Peter J. Cass (McK. '08), Guy D. Chandler (Ind. '11), Lewis W. Head (Chi. '11), John B. Johnson (McK. '10), Fred J. Lauman (K. C. V. C. '07), James P. Rimstidt (Cin. '13) and John E. Spindler.

# Address of Welcome*

*By* PROFESSOR C. BRESSOU, *Paris, France, Director of the Veterinary School at Alfort*

GENTLEMEN AND COLLEAGUES:

In the name of the College of Alfort, its teachers and students, I extend you a hearty welcome. We receive you at our school with sincere joy. Your coming gives us the rare opportunity of presenting cordial greetings to all of you and particularly to John R. Mohler, Chief of the United States Bureau of Animal Industry; and, further, it gives us the occasion to spend agreeable hours with distinguished confrères who are dear to us.

PROF. C. BRESSOU

Your presence here and the flattering terms announcing your visit in the tour program, terms I am pleased to recall: "Alfort, the famous veterinary school," are proofs of your high esteem. My colleagues appreciate this mark of respect and thank Dr. Eichhorn for the care he has taken in preparing this delightful occasion.

Alfort and the College of Lyon, its elder by two years, are the oldest veterinary schools in the world. It was here that Bourgelat made up the first rational curriculum of our art, from which fruitful thoughts were strewn in many directions. Concerning those closer to you, Vial de Saint-Bel was educated at Alfort before going to Great Britain, where he founded the London Veterinary College. Later, Liautard passed through Alfort before going to your great country, where his talent could expand, and where he established the American Veterinary College.

Your desire to become better acquainted with this cradle of the world's veterinary education is quite legitimate, although, except for the ground upon which the college stands, there are but few traces of the past. Alfort has gone through periods of evolution; it has followed the march of a science to which it has unceasingly contributed. You shall notice, however, some of the milestones of this progress. On the one hand, old buildings, ancient premises, where our forebearers spent many an hour of study, a testimony of their efforts; monuments erected to the memory of those who have attained a high reputation and in commemoration of their labors and their discoveries; on the other hand, new concepts, newer buildings better adapted to the needs of the scientific research and professional technics of our time. And so, we make a constant effort to honor the past and to uphold the traditions that have made us deserve your esteem, of which we are proud, and also to keep faith with the scientific culture and professional teachings founded by our predecessors.

Gentlemen, you are nearing the end of a long and fruitful journey. You have been in many countries and you have visited many educational institutions. You have met confrères of the entire world and already you see the time when you shall resume your daily tasks and family life. I wish that here, at the last stop of your journey, you already feel at home in a country which is close to yours, where our hearts have beaten as one during great conflicts of the past. We forget nothing of your generous sacrifices that would be prompted today by the same spirit when our common ideals are in danger.

Our wish will be fully satisfied if, during the few and too short hours you will be at Alfort, you would not only penetrate into our brotherly and hearty sentiments, but also feel the fervor of our friendship.

*Delivered upon the occasion of the visit of the members of the A.V.M.A. Official International Veterinary Congress Tour, to l'École Vétérinaire d'Alfort, September 5, 1938.

# Complement Fixation with Chick-Embryo Antigen in Equine Encephalomyelitis *

*By* WILLIAM M. MOHLER, *Washington, D. C.*
*Pathological Division, U. S. Bureau of Animal Industry*

The complement-fixation test is based on the discovery of Bordet and Gengou, in 1901, while investigating the nature of complementary substance of blood. This work led to other experiments upon antigens which enabled investigators to recognize and differentiate antigenic substances. It also resulted in the demonstration of antibodies in immune blood-serum. It was found that when a specific antibody and its complement are combined, they can attach themselves only to the antigen which produced that specific antibody. If the suspected serum contains the specific antibody, the antigen and complement unite with it. The complement is therefore fixed, so that no hemolysis occurs when the blood corpuscles and the hemolytic amboceptor are subsequently added.

These studies made possible the efficient diagnosis of many latent and obscure diseases in both man and animals. The method was adapted, in 1906, by Wassermann, Neisser and Bruck, to the practical diagnosis of syphilis of man and is popularly known as the Wassermann test. In veterinary medicine it has been successfully employed in the diagnosis of a number of diseases, particularly glanders and dourine. In fact, the practical elimination of both of these diseases in the United States is due largely to the reliability of the complement-fixation test in locating obscure and contact cases in their early stages.

In 1937, the complement-fixation test was used by Howitt,[1] of California, in studying experimental encephalomyelitis of guinea pigs by producing an antigen from the emulsified brain of injected guinea pigs and also mice. From these studies it was concluded that it is possible to differentiate the viruses of equine encephalomyelitis (western and eastern strains), Russian type encephalomyelitis and lymphatic choriomeningitis with such brain antigens.

Since the more recent outbreaks of this disease in this country, investigators[2, 3] have resorted to cross-immunity tests with guinea pigs and horses previously inoculated with eastern and western strains of formalinized vaccines or virus to differentiate these strains, using normal animals of susceptible species as controls.

The present investigation suggested itself because the new chick-embryo vaccine had been introduced about June, 1938, for the protective inoculation of horses as well as in the preparation of hyperimmune serum prepared by numerous inoculations with both horse-brain emulsions and chick-embryo vaccines of both strains of virus.

Attention had been focused on the fact that one chick embryo contained about 100,000 times as much virus as the brain of a laboratory animal and this large amount of virus was available in the space of about 18 hours from the time of inoculation. Knowing these facts to be definitely conclusive, it was decided to inoculate eleven-day-old chick embryos with western type virus recovered from guinea-pig brains. These embryos were then used in the preparation of an antigen for the complement-fixation test.

A 20 per cent emulsion of guinea pig-brain virus in normal saline solution was inoculated into the eggs containing live embryos, after very small windows or openings had been prepared through the shells. In the preparation of the embryo eggs for inoculation with the virus, every precaution is taken to prevent the possibility of contamination. After the eleven-day-old embryos have been inoculated with the virus, the opening is covered with sealing

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paper or wax and the eggs are replaced in the incubator, which is regulated to 100° F.

After further incubation for 18 hours, the eggs are opened and the embryos removed in a sterile manner. Some of the embryo virus is retained for the subsequent passage into normal chick embryos and also for sterility tests and guinea pig inoculations. The embryo virus is placed in a sterile mortar and ground up into an emulsified mass. To one part of embryo virus, two parts of distilled water are added and the mixture is again ground thoroughly. This mixture is then transferred from the mortar into eight-ounce bottles with cork stoppers and placed on the shaking-machine for about one hour. This material is then placed in the centrifuge for about 15 minutes at 2,500 revolutions per minute. The supernatant fluid is poured off and placed in the ice-box to be used as a stock antigen.

In applying the complement-fixation test, each antigen is titrated beforehand. At the present time, the above dilution with distilled water has been sufficient, but these antigens have demonstrated such strong reactions against complement-binding serums that it would be possible to make further dilutions if the quantity of stock antigen were low.

Titrations of the antigens were begun immediately after each of a series of passages of the embryo virus had been prepared as above. The antigens were titrated against a known normal horse serum and a known hyperimmune equine encephalomyelitis serum, both of which had been previously inactivated at 58° C. for 35 minutes in a water-bath. Although it was determined in these tests applied to the first six consecutive passages of chick-embryo-virus antigens that these antigens demonstrated some antigenic value against a complement-binding serum, it was not possible to reach any definite conclusions until the seventh passage of chick-embryo-virus antigen was tested. At about this time, six serums became available from horses previously vaccinated or immunized by inoculations with formalinized horse-brain-virus emulsions, chick-embryo vaccine (western and eastern strains), or both, at varying intervals.

The reactions of this chick-embryo-virus antigen, seventh passage, against the suspected positive horse serums and a normal horse serum for control are shown in table I.

Titrations by the complement-fixation test were applied to 21 consecutive passages

TABLE I—*Antigen titration against suspected positive horse serums.*

| ANTIGEN (CC) | 0.1 | 0.2 | 0.4 | 0.6 | 0.8 | 1.0 | 1.5 | NORMAL SERUM CONTROL | ANTIGEN CONTROL |
|---|---|---|---|---|---|---|---|---|---|
| Normal serum (0.2 cc) | — | — | — | — | — | — | — | — | — |
| ANTIGEN (CC) | 0.05 | 0.1 | 0.2 | 0.3 | 0.4 | 0.5 | 0.8 | 1.0 | P. S. CONTROL (NO ANTIGEN) |
| S. P. S. 1 (0.2 cc) | — | + | 2+ | 4+ | 4+ | 3+ | 2+ | + | — |
| S. P. S. 2 (0.2 cc) | + | 4+ | 2+ | + | + | + | + | + | — |
| S. P. S. 3 (0.2 cc) | — | — | + | 2+ | 4+ | 4+ | 4+ | 4+ | — |
| S. P. S. 4 (0.2 cc) | — | — | 2+ | 3+ | 4+ | 2+ | + | + | — |
| S. P. S. 5 (0.2 cc) | — | — | — | — | — | — | — | — | — |
| S. P. S. 6 (0.2 cc) | 4+ | 4+ | 4+ | 4+ | 4+ | 4+ | 4+ | 4+ | — |

P. S.=positive serum.
S. P. S.=suspected positive serum.
—=complete hemolysis.
+=slight inhibition of hemolysis.
2+=partial inhibition of hemolysis.
3+=moderate inhibition of hemolysis.
4+=complete inhibition of hemolysis.

Serums 1 to 5, inclusive, were drawn from horses receiving formalinized horse-brain virus of the western type for the production of commercial curative serum. Serum 5 shows a lack of complement-fixing bodies.

Serum 6 was drawn from horses receiving chick-embryo vaccine (western and eastern types).

of the chick-embryo-virus antigens against our stock complement-binding serum and all of these antigens demonstrated exceptional antigenic value with the exception of the first six passages, which have been previously mentioned.

After numerous tests with chick-embryo-virus antigens had been applied against known complement-binding serums, some of which were known to have been drawn from horses immunized with formalinized chick-embryo vaccine, the question arose as to whether these strong fixation reactions with small amounts of the antigen were being brought about by the presence of protein substances in the tissues of the embryos. Consequently, a tissue emulsion was prepared from a number of eleven-day-old normal chick embryos, using the same technic as followed with our virus antigens.

The results of tests with normal chick-embryo emulsion are given in table II.

Table II indicates that normal horse serum gave a complete hemolysis to normal chick-embryo protein. Similar negative results were obtained with the positive serum from one horse (B) that received horse-brain virus intralingually and from three horses (D, E, and F) that had recovered spontaneously without serum or chick-embryo vaccine. On the other hand, the two positive cases (A and C) had received numerous inoculations of chick-embryo vaccine and virus to increase the potency of the serum, horse C having been inoculated with large quantities of this product weekly for over three months, while horse A had received a smaller amount. The fact that the former showed such a marked reaction may be due to the large amount of chick protein which was present in the inoculant.

Further proof that embryo protein, *per se*, is not responsible for these strong fixation reactions is shown by the fact that rather indefinite results were obtained with the first six consecutive passages of chick-embryo-virus antigens, but the seventh and subsequent passages of this virus through chick embryos produced strong fixation reactions with minute amounts of antigen.

Recently, in connection with these studies on the complement-fixation test, it was decided to obtain samples of serum from horses which had recovered from an attack of equine encephalomyelitis and in which no specific treatment had been given, that is, no serum or vaccine, and also from horses that had been in intimate contact with cases of encephalomyelitis but had not shown any clinical evidence of the disease.

Upon request, Dr. G. W. Cronen, inspector-in-charge at Helena, Montana, forwarded 20 samples of horse serums drawn from animals which had been previously affected and had recovered, and also from apparently normal animals which had been in intimate contact with these infected ani-

TABLE II—*Normal chick-embryo protein emulsion titration against positive horse serums.*

| EMULSION (CC) | 0.1 | 0.2 | 0.4 | 0.6 | 0.8 | 1.0 | 1.5 | NORMAL SERUM CONTROL | ANTIGEN CONTROL |
|---|---|---|---|---|---|---|---|---|---|
| Normal serum (0.2 cc) | — | — | — | — | — | — | — | — | — |
| EMULSION (CC) | 0.05 | 0.1 | 0.2 | 0.3 | 0.4 | 0.5 | 0.8 | 1.0 | P. S. CONTROL (NO ANTIGEN) |
| P. S. A (0.2 cc) | — | — | — | — | 3+ | 4+ | 4+ | 4+ | — |
| P. S. B (0.2 cc) | — | — | — | — | — | — | — | — | — |
| P. S. C (0.2 cc) | — | 4+ | 4+ | 4+ | 4+ | 4+ | 4+ | 4+ | — |
| P. S. D (0.2 cc) | — | — | — | — | — | — | — | — | — |
| P. S. E (0.2 cc) | — | — | — | — | — | — | — | — | — |
| P. S. F (0.2 cc) | — | — | — | — | — | — | — | — | — |

P. S.=positive serum.
—=complete hemolysis.
3+=moderate hemolysis.
4+=complete inhibition of hemolysis.

mals during the progress of the disease. Four additional samples of serum from horses in Oklahoma, which were collected over 16 months ago, were utilized for test purposes. These four serum samples were drawn from horses, three of which were in contact with the disease, and all three gave a negative reaction to the test. The remaining serum sample had been drawn from a horse which had recovered from an attack of encephalomyelitis two weeks previous to the collection of the serum. When tested, this serum gave a 4+ reaction against the western antigen.

After several tests had been applied to the serum samples received from Montana and also to samples drawn from horses used in our experimental work at Beltsville, Maryland, it was decided to retest these serums against the western chick-embryo-virus antigen and the normal chick-embryo emulsion. Previous to testing these serums, a titration was made of both antigen and normal chick-embryo emulsion, using the regular routine procedure with controls for each consecutive step of the test. In this titration, as in all titrations in the complement-fixation test, the smallest amount of antigen which completely fixed the complement, in the presence of the complement-binding serum, was the antigenic unit. The antigenic dose of both antigen and chick-embryo emulsion used in the test proper was double the antigenic unit.

TABLE III—*Results of tests with serums against virus antigen and normal chick-embryo emulsion.*

| HORSE | LOCATION | HISTORY | RESULTS AGAINST WESTERN CHICK-EMBRYO ANTIGEN | RESULTS AGAINST NORMAL CHICK-EMBRYO EMULSION |
|---|---|---|---|---|
| 1 | Montana | Affected, July, 1938; recovered | 4+ | — |
| 2 | | Contact | — | — |
| 3 | | Affected, July, 1938; recovered | 4+ | — |
| 4 | | Contact | — | — |
| 5 | | Affected, July, 1938; recovered | 4+ | — |
| 6 | | Contact | — | — |
| 7 | | Contact | — | — |
| 8 | | Contact | — | — |
| 9 | | Contact | — | — |
| 10 | | Contact | — | — |
| 11 | | Contact | — | — |
| 12 | | Affected, July, 1938; recovered | 4+ | — |
| 13 | | Affected, July, 1938; recovered | 4+ | — |
| 14 | | Contact | — | — |
| 15 | | Contact | — | — |
| 16 | | Contact | — | — |
| 17 | | Contact | — | — |
| 18 | | Contact | — | — |
| 19 | | Contact | — | — |
| 20 | | Affected, July, 1937. No serum or vaccine used; no disease or treatment in 1938 | 4+ | — |
| 21 | Beltsville, Maryland | Two inoculations intralingually with western virus, March, 1938 | 4+ | — |
| 22 | | Vaccinated several times with western vaccine; two injections with eastern virus | 4+ | — |
| 23 | | One injection western virus several days before test | + | — |
| 24 | | Immune to eastern virus; result of recovery from disease | — | — |

—=complete hemolysis.
+=slight inhibition.
4+=complete inhibition.

Sample 24 was positive to eastern antigen, but negative to western antigen. This horse had been inoculated previously with an eastern strain of virus intralingually and showed definite symptoms, but made a recovery. At a later date, this animal was again inoculated intracerebrally with an eastern strain of virus and remained normal. It had not received any inoculations of chick-embryo vaccine at any time.

This report has dealt chiefly with the use of the western chick-embryo-virus antigens; however, a number of tests against eastern complement-binding serums with a formalinized eastern chick-embryo-vaccine antigen demonstrated that these serums gave a 4+ reaction or complete inhibition of hemolysis, whereas these serums, when tested against a western chick-embryo-virus antigen, gave a negative reaction or complete hemolysis, and vice versa. The formalinized antigens, however, did not show so wide a range between the antigenic unit and the anticomplementary point as did the nonformalinized material.

The formalinized eastern chick-embryo antigen in the various titrations against eastern complement-binding serum has demonstrated a certain degree of specificity and a further report on tests with both strains of equine encephalomyelitis chick-embryo-virus antigens non-formalinized will be made at a later date.

As previously indicated, these tests show the presence of antibodies in recently immunized horses, which naturally leads to the possibility of its use as an aid in determining the potency of curative serum produced by hyperimmunizing horses by those biological houses preparing this product.

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## No Rabies Deaths in Chicago During 1938

According to a report issued December 27, by the Chicago Board of Health, no deaths from rabies occurred in the past year, although 14,904 people were bitten by dogs. This is the second successive year that the city has reported no rabies deaths.

## Visitors at the Journal Office

An unusually large number of veterinarians have visited the JOURNAL office during recent months. Among the number who have called on various missions are the following: Drs. H. D. Bergman, of Ames, Iowa, president of the A. V. M. A.; M. Jacob, of Knoxville, Tenn., treasurer of the A. V. M. A.; J. R. Mohler, J. E. Shillinger and Lt. Col. Raymond A. Kelser, of Washington, D. C.; Drs. L. M. Hurt, of Los Angeles, Calif.; J. L. Cherry, of Christiansted, Virgin Islands; D. M. Warren, of Missoula, Mont.; W. T. Oglesby, of Baton Rouge, La.; R. A. Runnells, of Ames, Iowa; R. H. Lay, of Winnipeg, Man., Can.; T. A. Sigler, of Greencastle, Ind.; W. M. Swangard, of Vancouver, B. C.; S. E. Hershey and H. M. Newton, of Charleston, W. Va.; J. V. Lacroix, of Evanston, Ill., and A. C. Merrick, of Brookfield, Ill.

## Tularemia Epidemic in Illinois

In the week ended December 10, there were 128 new cases of tularemia in Illinois —more than double the number for the preceding week—according to a report from the State Department of Health. Ten deaths from the fever have been reported during the current season.

Blood transfusions from persons recently recovered from the disease have been found to be the most effective treatment, but due to the scarcity of such donors, control of the epidemic has become a difficult problem.

## Bureau Transfers

DR. LOWELL R. BARNES (O. S. U. '35), from San Juan, Puerto Rico, to Indianapolis, Ind., on tuberculosis eradication.

DR. ARTHUR G. BEAGLE (K. C. V. C. '18), from Mason City, Iowa, to Sioux Falls, S. Dak., on meat inspection.

DR. JOHN S. GWALTNEY (Ind. '21), from Trenton, N. J., to Montpelier, Vt., on tuberculosis eradication.

DR. HARRY E. KEMPER (K. C. V. C. '14), from Bismarck, N. Dak., to Albuquerque, N. Mex., on field inspection and tuberculosis eradication.

DR. WM. R. RICHARDS (Chi. '08), from Watertown, S. Dak., to Louisville, Ky., in charge of meat inspection.

DR. OTTO J. SCHRAG (Iowa '26), from Sioux City, Iowa, to Sioux Falls, S. Dak., on meat inspection.


## FLUKES IN THE RESPIRATORY TRACT OF DUCKS*

*By* F. R. BEAUDETTE, *New Brunswick, N. J.*
*New Jersey Agricultural Experiment Station*

On September 28, 1936, a live Mallard duck from a lake in Gloucester County, New Jersey, where some losses were reported, was submitted for examination. Although respiratory symptoms were not noticed, three flukes were found in the upper part of the trachea. These parasites were red. The mucosa was not altered. There were ulcerations in the gizzard and several nematodes were found beneath the cuticula. These were diagnosed as *Epomidiostomum uncinatum* by Dr. E. E. Wehr, to whom thanks are due. Finally, several firmly attached acanthocephalids were found in the posterior third of the small intestine. Thanks are due to Dr. B. G. Chitwood for the diagnosis of these as *Profilicollis botulus*.

On October 27, 1936, two more live ducks were brought in from a lake in Gloucester County. One of these had an injury of the neck and on autopsy showed hepatitis. A blood smear showed anemia. The other duck was in good flesh and autopsy revealed a fluke just inside the larynx and another at the bifurcation of the trachea. These flukes also were red. The parasite remained in one place, but continuous waves of contractions of the body were seen. This bird also was infested with acanthocephalids.

Finally, on August 10, 1937, three dead wild ducks from a lake at Avon, Monmouth County, New Jersey, were brought in. According to the history, the birds developed limber neck, and eight or ten had died. The case was diagnosed as botulism, but in the upper part of the trachea of a decomposed bird a single fluke was found.

Flukes from the three cases were submitted to Dr. H. W. Stunkard, of the Department of Biology, New York University, who diagnosed them as *Typhlocoelum cymbium* (Diesing, 1850) Kossack, 1911. Here I wish to express my appreciation to Dr. Stunkard.

This species was first reported in North America in 1928, by Manter and Williams,[1] at Lincoln, Nebraska. Out of 13 Pintail ducks, *Dafila acuta* (L.), three were infested. However, these authors refer to it as *Tracheophilus sisowi* Skrjabin 1913, but as Stunkard[2] points out, there is no apparent difference between this and *T. cymbium*.

The life cycle of this parasite has been worked out by Stunkard,[2] who began with a single specimen from the nasal passages of a pied-billed grebe (*Podilymbus podiceps*). Three species of snails, *Helisoma trivolvis, Lymaea stagnalis* and *Physella heterostropha*, were exposed to free-swimming miracidia, but only two of the three Helisoma were found to be infested on dissection. Unfortunately, a domestic duck was not found to be infested twelve weeks after a feeding of six cysts.

These cases are recorded so that poultry pathologists will look for similar ones, for, as Stunkard points out, the species is cosmopolitan and enjoys wide distribution because of the migratory host and the fact that larvae develop in common snails.

*Journal Series paper of the New Jersey Agricultural Experiment Station, department of poultry husbandry. Received for publication, January 8, 1938.

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# CONGENITAL HYPERTROPHIC KERATINIZING DERMATOSIS IN A CALF*

*By* G. L. DUNLAP, *Urbana, Ill.*
*Laboratory of Animal Pathology and Hygiene, University of Illinois*

This specimen was presented at the laboratory, for diagnosis, from the practice of Dr. H. L. Keene, Shabbona, Ill., with the following history:

> I am sending under separate cover specimen of hide of a new-born calf delivered this morning. The same condition existed in a calf born ten days ago in the same purebred Brown Swiss herd. When born, there appeared to be no hair, but when dried the surface cracked and exposed the hair underneath. As the skin dried, blood exuded from the cracks of the hide. When these calves were delivered, this was a rubbery substance and of a yellow color, but as it dried, it turned white. The entire hide of the calf is like the sample. Both calves were born alive and fully developed but were never able to stand. The herd is in perfect health, under full feed, and in ideal condition. No abnormalities have ever been observed in the herd. Both calves are by the same sire (ten years old) and the dams of the affected calves are mother and daughter. In one case it is a first calf, while in the other the cow has produced five healthy calves.

*Received for publication, March 19, 1938.

*Gross lesions:* The specimen received is shown in figure 1, which is about two-thirds natural size. The hide measures 1.5 cm in the thickest part. The surface is dry and rather hard and presents an appearance resembling the rough bark of a tree. There are irregular transverse and longitudinal cracks in the surface. The hair shafts are exposed in these cracks, giving the hide the appearance of a "felt mat."

*Microscopic lesions:* Hematoxylin and eosin-stained sections show a markedly hypertrophic epidermis. The deep layers and the subcutis are normal except that the connective tissue fibers appear coarser than normal. The layers of the epidermis are not distinct. The cornifying layer shows a marked overgrowth. This material stains pink with hematoxylin and eosin, and yel-

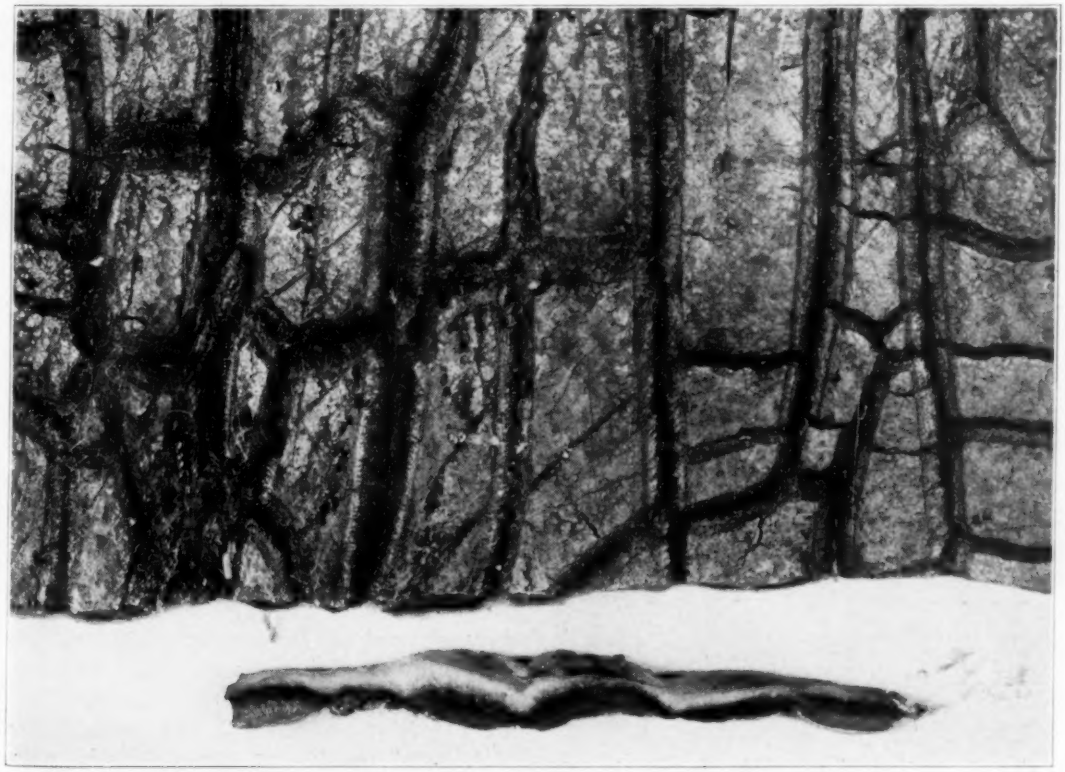

FIG. 1. Calf hide. Two-thirds natural size. Cross-section below.

low with Van Gieson's mixture, showing that it is of epithelial hyaline in nature. Cell outlines in this hyaline mass are visible but there is no nuclear staining. Hair shafts are abundant (fig. 2).

This condition is apparently a disturbance of development and probably may be traceable to close inbreeding in the herd, though no data on the hereditary factor or factors involved are established in this case.

*Diagnosis:* Congenital hypertrophic keratinizing dermatosis.

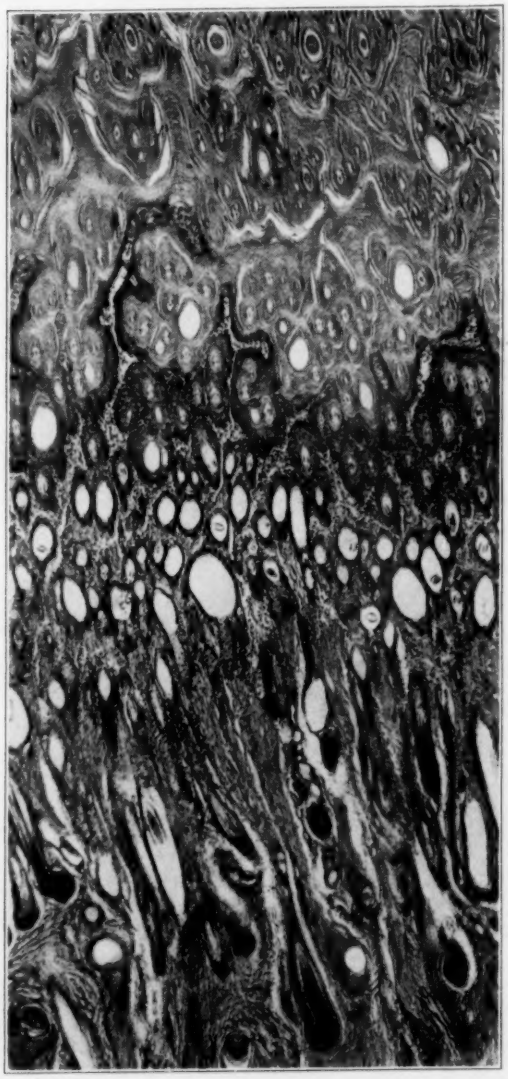

FIG. 2. Section of calf hide showing numerous hair-shafts, indistinct epidermal layers and excessive cornification (x 40).

## POISONING IN CATTLE TRACED TO ARSENIC IN GRASSHOPPER BAIT*

*By* G. L. DUNLAP, *Urbana, Ill.*
*Laboratory of Animal Pathology and Hygiene, University of Illinois*

The subjects for this report were submitted from the practice of Dr. H. F. Jones, of Hoopeston, Ill. Two five-month-old calves, one dead and one sick, were presented at the laboratory for examination.

*History:* A herd of 13 purebred and grade Angus cattle of different ages had access to a stalk-field during the day and were placed in a lot with a shed for shelter during the night. There had been a loss of four calves prior to the time the two specimens were presented at the laboratory. The live calf, according to the attending veterinarian and owner, was a typical clinical case. The dam of the calf also was affected. Onset of the symptoms was sudden and the course of the illness lasted three to four days before death. All animals in the herd were inoculated with pulmonary mixed bacterin.

FIG. 1. Calf suffering from "grasshopper bait" poisoning, showing weakness, unsteadiness, and "tucked up" abdomen. Calf died a few minutes after picture was taken.

*Symptoms:* The calf (fig. 1), on arrival at the laboratory as well as the following day, showed a temperature of 103° F. Breathing was labored and rapid. The animal displayed a "tucked up" abdomen which, on palpation, was rigid and tense. The calf stood quietly or moved slowly with

*Received for publication, March 19, 1938.

caution, evincing symptoms of pain. Inappetence, great thirst, and a watery diarrhea were observed in the patient and were reported as symptoms in all affected animals. There was some drooling of saliva.

*Blood examination:* Hemoglobin, 15.08 gm per 100 cc (Newcomer); red blood-cells, 4,860,000 per cmm; white blood-cells, 5,350 per cmm; polymorphonuclears, 45 per cent; lymphocytes, 50 per cent; mononuclears, 4 per cent, and eosinophiles, 1 per cent.

*Bacteriological examination:* Jugular blood drawn aseptically was introduced freely on plain and liver agar plates and in meat mash media. The inoculated media, incubated at 37.5° C. for 48 to 72 hours, remained sterile. A rabbit, a guinea pig and a pigeon were each inoculated with 2.0 cc of jugular blood, with no clinical ill effects.

*Fecal examination: Ostertagia* eggs were easily demonstrated in the feces by the concentration method.

*Urine examination:* Albumin, positive; sugar, bile and hemoglobin, negative; ketones, slight trace; reaction to litmus, acid. Microscopic examination of sediment of centrifuged sample showed many epithelial cells.

*Autopsy:* The calf died 48 hours after arrival at the laboratory. Examination showed a well developed and well nourished calf of the stated age of five months. On opening the abdominal cavity, a large quantity of slightly bloody, slightly clouded, yellowish, foul-smelling liquid escaped. There was a fibrino-purulent exudate covering the parietal and visceral peritoneum with early, easily broken down adhesions. Careful examination of the peritoneal surface of the intestinal tract showed no perforations. Both surfaces of the liver and spleen showed a fibrino-purulent exudate, while the parenchyma of both organs was normal except for congestion. The heart was normal. Lungs were a bright pink color with smooth glistening surfaces, while palpation as well as cut section revealed no areas of pneumonia.

Examination of the abomasum showed very little ingesta. The contents were carefully washed and examined for parasites but none were found. The mucous mem-

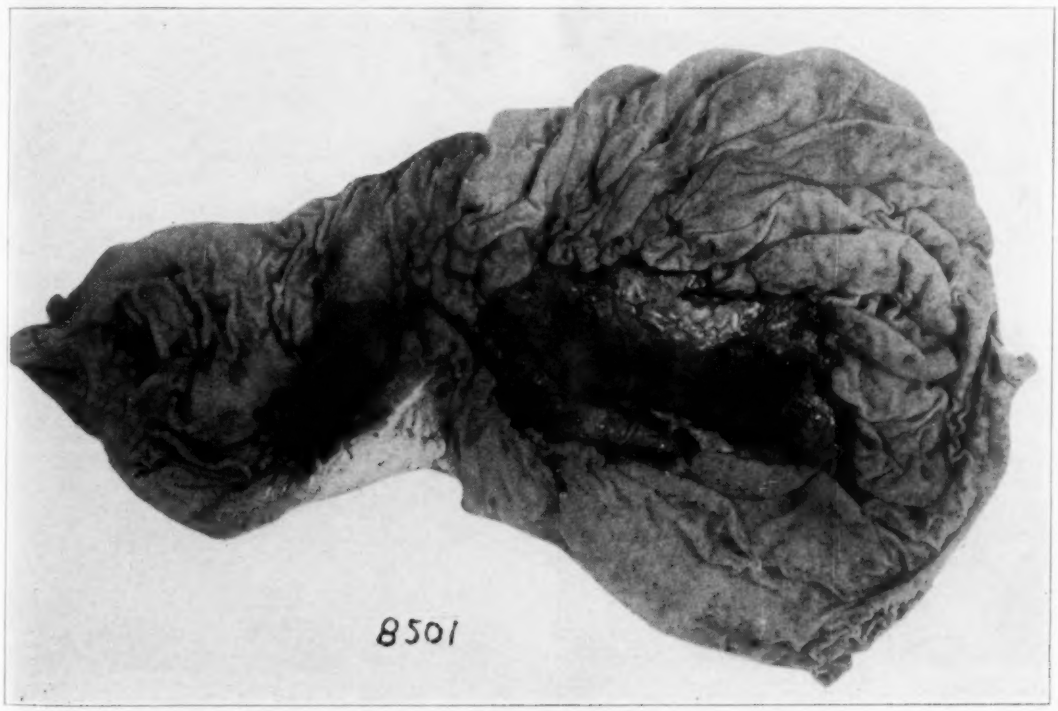


FIG. 2. Abomasum, showing the two large, denuded areas and thick edematous folds of the mucous membrane.

brane was thrown up in large, thick, edematous folds. The surface presented two large, sharply circumscribed, denuded "ulcers." The larger of these denuded areas measured 3 x 4 inches. Toward the pyloric end of the abomasum, there were several smaller erosions. The mucous membrane in these areas was eroded to the muscular coat. See figure 2. The fore-stomachs, as well as the small and large intestines, were essentially normal.

The second calf, which was dead on arrival, had shown similar symptoms. Advanced postmortem decomposition was encountered, although lesions in the abomasum and peritoneum were analogous to those described.

*Histopathology:* Sections from the involved areas of the abomasum showed an extensive necrosis involving all the structures in the wall of the organ. The mucous membrane was denuded over much of the area, so that the inner muscular layer was in direct contact with the ingesta. The mucous membrane that remained (fig. 3) was ill defined and most of the cells showed no nuclear staining. The submucosa showed marked edema, while the muscular coats showed varying degrees of necrosis in different fields. The striking feature of this lesion was the absence of a cellular inflammatory reaction in its base. However, the margins of the lesions, between the dead and living tissue, showed a zone of inflam-

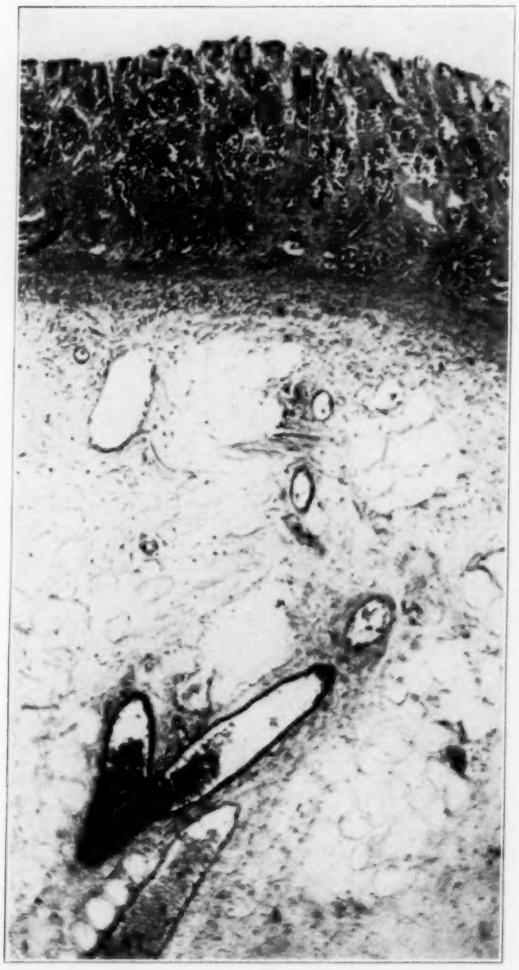

FIG. 3. Section of wall of abomasum near margin of involved area. Architecture retained but very little nuclear staining (x 70).

FIG. 4. Section of wall of abomasum at margin of lesion showing cellular reaction between dead and living tissue (x 70).

matory cells (fig. 4). Large colonies of bacteria, presumably of intestinal origin, were present in the mucosa, submucosa, and muscular coats of the involved areas, and in the fibrinous exudate on the peritoneal surface.

The presence of the rapidly necrotizing process of the epithelium and deeper structures of the abomasum without any cellular defense reaction suggested a strong caustic substance as the probable cause. A search for a possible source of poison to which the herd had access was initiated and it was learned that grasshopper bait had been stored in the feed-trough of the cattleshed. Evidence that it had been consumed by the cattle was verified by the owner and by chemical examination.

*Chemical examination:* A chemical examination of the liver for arsenic by Dr. H. H. Mitchell, Professor of Animal Nutrition, University of Illinois, disclosed 1.12 mg of arsenic trioxide per kg of fresh tissue. This amount is approximately ten times greater than that reported as normal for human tissues.

*Diagnosis:* Arsenic poisoning.

*Acknowledgment:* Assistance in the examination of these specimens was given by Drs. Robert Graham, J. Sampson and L. E. Boley, of the laboratory staff.

## NEUROLYMPHOMATOSIS PHASIANORUM*

*By* ERWIN JUNGHERR, *Storrs, Conn.*
*Department of Animal Diseases, Storrs Agricultural Experiment Station*

The purpose of the present communication is to record the occurrence of a disease in ring-necked pheasants (*Phasianus torquatus* Gmelin) which resembles fowl paralysis (neurolymphomatosis gallinarum) in clinical and histologic manifestations.

The outbreak occurred in a commercial pheasant flock of about 1,000 four-month-old birds, which were kept in out-door cages and fed a standard turkey mash fortified with additional cod-liver oil. According to the owner's statement, no other species of domesticated or game birds were kept on the premises.

The disease affected about 3 per cent of the flock and was characterized by paralysis of the legs in various degrees, and occasionally by symptoms of "wry neck."

Of six live pheasants submitted for laboratory examination, one was killed immediately and the five others kept for an observation period of ten days, at the end of which they were sacrificed for gross and histologic examination. A cultural study made at the same time failed to reveal the presence of significant organisms.

The first specimen did not show clinical paralysis. At autopsy, although in good flesh and otherwise normal, the muscles of the leg in the region of the tibialis anticus and ambicus and vastus internus exhibited grayish-yellowish cross striations. The lesions appeared to be degenerative in character and suggested a nutritional origin of the disease.

To test this possibility, two birds were placed on a turkey mash plus 15 per cent Wilson's vacuum-dried liver meal. This supplement was shown to be rich in the *chick-antiparalytic* factor ($B_4$) by Keenan *et al*,[1] and from previous tests the particular lot was known to be curative for field or experimental cases of $B_4$ deficiencies in chicks within one to three days. Two other birds were placed on the same mash plus 5 per cent raw soybean oil. The latter supplement is a preventive of muscle degeneration in ducklings which are fed a diet lacking the *chick-anti-encephalomalacic* factor (Pappenheimer and Goettsch[2]). The lot of soybean oil used was known to be effective in the prevention of experimental encephalomalacia of chicks. No improvement in the symptoms could be noted in either lot, during the period of observation. The remaining bird was clinically affected with "bumblefoot" and regarded as a control.

### HISTOLOGIC STUDY

Blocks of tissues were fixed in non-acidified Zenker's fluid or in Bouin's for the nervous organs, embedded in paraffin and

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stained by ordinary methods. During the period of observation, the birds were subjected to routine hematologic examinations, without finding any conspicuous alterations in the blood-picture, except in the bumblefoot case, which showed a significant heterophile leukocytosis. A summary of the findings is presented in table I.

The lesions of the voluntary muscles (fig. 1) were characterized by extensive myodegeneration in the affected areas. In cross-section the Cohnheim fields of the muscle fibers showed either granular alterations or replacement of the normal tissue by hyaline or waxy material. Longitudinal sections exhibited the characteristic appearance of Zenker's degeneration. Probably the most conspicuous alteration was brought about by the apparent proliferative activity of the perimysium proper. In certain areas the bundles seemed to be widely separated, partly due to edema, partly due to an increase of fibroblastic and histioblastic elements. In the borderland between healthy and diseased muscle, the normally oblong fibrocytes appeared vesicular and spherical, with occasional mitotic figures testifying to the pathologic activity. Here and there syncytial clumps of fibroblasts were situated in bays of degenerated muscle fibers, which suggested a resorptive process. Some proliferating cells showed the spongy nucleus of histiocytes, but true granulocytes were rarely in evidence.

The lesions in the other organs were of the small-cell mononuclear type characteristic of neurolymphomatosis. The pancreas failed to show evidence of infiltration, which would be in line with the experience in chicks that this organ is rarely implicated in neurolymphomatosis, in contrast with epidemic tremor, in which it is almost constantly affected. Stimulation of the lymphopoietic system was seen in the liver, kidneys (fig. 2) and spleen (fig. 3). The central nervous system showed similar focal lesions varying from perivascular infiltrations (fig. 4) to glia nodules and whorl-like cicatricial changes. In two cases the sciatic nerves (fig. 5) showed lymphoid infiltrations, which are considered to be pathognomonic for neurolymphomatosis in the common fowl.

TABLE I—*Histologic lesions* of neurolymphomatosis phasianorum.*

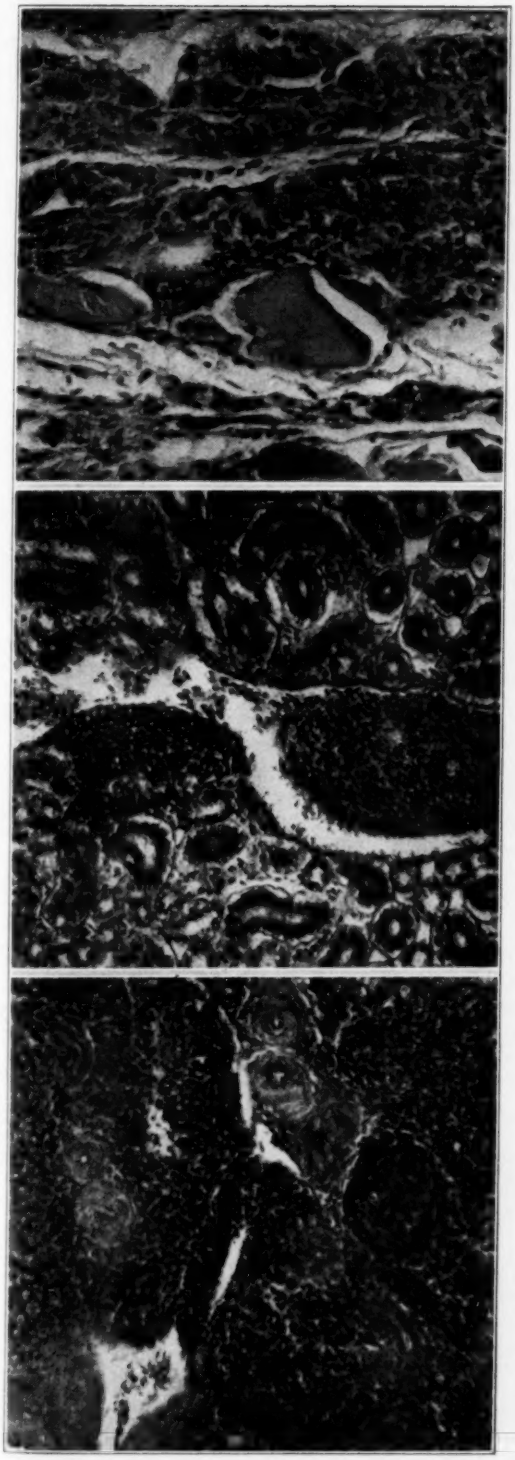
| Specimen | Clinical Paralysis | Experimental Treatment | Histological Findings | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Leg Muscle | Liver | Spleen | Kidney | Gizzard | Sciatic Nerve | Cerebellum | Optic Lobe | Cerebrum |
| 1 | — | | +++ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 2 | ++ | 15% liver meal ineffective | — | — | ++ | + | — | + | — | + | ++ |
| 3 | + | | +++ | — | + | + | — | — | ++ | ++ | +++ |
| 4 | +++ | 5% soybean oil ineffective | — | + | — | — | + | ++ | + | — | + |
| 5 | + | | — | + | — | — | — | — | ++ | + | +++ |
| 6 | Bumblefoot | Control | — | — | — | — | — | — | — | — | — |

*Monocytic foci, except in muscle. 0 = not examined.

## DISCUSSION

The occurrence of the syndrome of neurolymphomatosis in pheasants raises three important questions, namely, those of diagnosis, nomenclature, and relationship to the fowl paralysis problem as a whole.

The diagnosis was based upon (1) clinical symptoms, (2) failure to demonstrate a microbial cause of the condition, (3) failure to observe improvement after feeding concentrates of potent avian antiparalytic factors, (4) histologic demonstration of characteristic lesions in the parenchymatous and nervous organs, and (5) failure to find similar lesions in a control bird. With the exception of myodegeneration of the striated musculature, which is not within the usual range of morphologic manifestations of fowl paralysis, the condition in



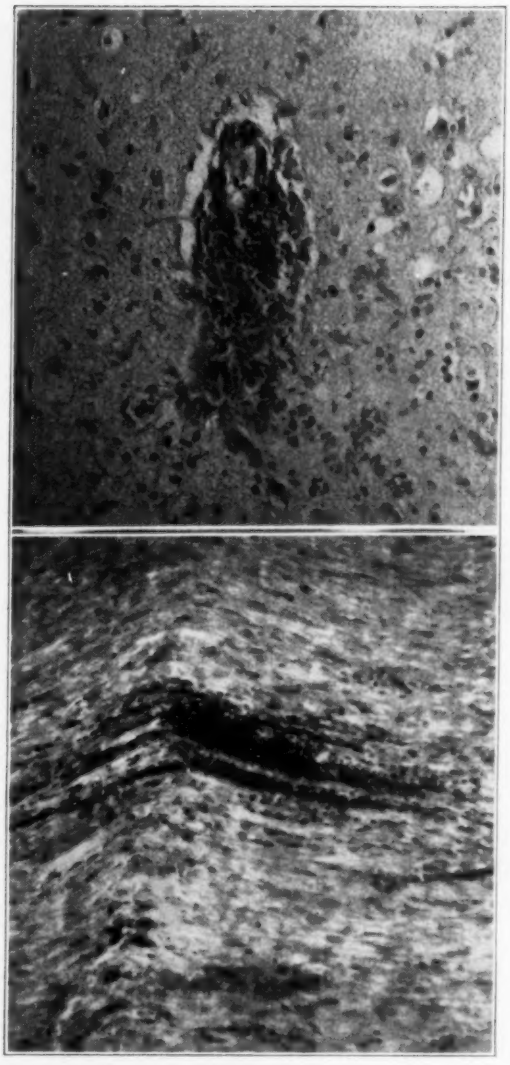


FIG. 1 (upper left). Cross-section of M. tibialis anticus, showing waxy degeneration and proliferation of perimysium (x 160).

FIG. 2 (center left). Kidney showing a lymphoid focus adjacent to a large vessel (x 160).

FIG. 3 (lower left). Spleen showing a hypertrophic Malpighian corpuscle and lymphoid hyperplasia in the pulp (x 160).

FIG. 4 (upper right). Section of cerebrum in the region of the corpus striatum showing a perivascular focus (x 160).

FIG. 5 (lower right). Section of sciatic nerve showing lymphoid infiltration characteristic of neurolymphomatosis (x 160).

pheasants is pathologically indistinguishable from neurolymphomatosis gallinarum.

To apply this term to the condition in pheasants is not justified until the etiologic identity of the disease in the respective hosts can be established. Until this is done, the term *neurolymphomatosis phasianorum* is suggested.

As has been pointed out by the author,[3] until a few years ago, species-specificity was considered to be an outstanding feature of filtrable-agent diseases of the common fowl. Recent investigations by Jármai[4] and others, however, have shown that pheasants, poults and guinea fowl are susceptible to the erythroleukosis agent of the chicken. The present report furnishes evidence regarding the occurrence of a related pathologic entity, namely, neurolymphomatosis, outside of the ordinary zoölogic host. On the other hand, the apparent rarity of the disease in pheasants may point toward this species as a valuable animal in transmission trials in fowl paralysis, one important obstacle to which has been the spontaneous occurrence of the disease in ordinary domestic fowl.

SUMMARY

The occurrence of a fowl paralysis-like condition in a flock of 1,000 four-month-old, ring-necked pheasants (*Phasianus torquatus* Gmelin) is reported. Until the etiologic identity of the chicken and pheasant disease can be established, the term *neurolymphomatosis phasianorum* is proposed. The diagnosis was based upon ineffectiveness of treatment with chick-antiparalytic factors ($B_4$ and anti-encephalomalacic) and upon the demonstration of histologic lymphomatotic lesions in the parenchymatous organs and the peripheral and central nervous systems. Some cases were associated with myodegeneration and perimysial proliferation, in the striated muscles. The findings suggest absence of species-specificity of neurolymphomatosis and the possibility of using pheasants in experimental transmission of fowl paralysis.

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## ONION POISONING IN HORSES*

*By* FRANK THORP, JR. *and* G. S. HARSHFIELD, *Colorado Experiment Station, Fort Collins, Colo.*

In 1909, Goldsmith[1] reported the occurrence of onion poisoning in nine head of cattle which had eaten sprouted and decayed onions. The onions had been discarded in a pasture where animals were grazing. A week later, the owner found one cow dead and the remainder ill. The symptoms observed were an intense onion odor, constipation in some, slight purgation in others, and dark-colored urine which smelled of onions.

On postmortem examination of the dead animal, the following changes were observed: inflammation of the rumen; patchy inflammation of the intestines; a light-colored, enlarged liver; kidneys dark green in color and having an offensive odor. The rumen contained large quantities of onions and grass, and the carcass smelled of onions.

Pipal[2] observed symptoms of poisoning among healthy cattle that had been removed from a short pasture to a woods pasture where they grazed heavily on wild onions.

Klimmer[3] mentions onions among the poisonous plants, listing gastroenteritis, constipation, moderate diarrhea and vomiting as symptoms in cattle.

In 1932, Newsom[4] observed poisoning among cattle that had eaten frozen onions. The most outstanding symptoms were the odor of onions on the breath and coffee-colored urine. In this outbreak seven or eight head of cattle died.

Durrell and Newsom[5] mention several outbreaks of onion poisoning in cattle, followed by serious losses. The losses occurred late in the fall, among cattle run-

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ning on fields after onions had been dug and some left on the ground. The outstanding symptom was blood-stained urine, and on autopsy the body tissues smelled of onions.

Dr. H. L. Franklin informed the Colorado Experiment Station of an outbreak of onion poisoning among horses on a farm near Greeley, Colo., during January, 1938. Nine horses had been pastured over the entire farm all winter. They had access to an onion field where cull and unharvested onions were present. The outbreak occurred following a period of alternate freezing and thawing. Partly consumed onions were observed in the field.

All animals sickened within a period of 24 hours and, during that time, three died. Two more had died at the time of the investigation the next day. Dr. Franklin later reported two more deaths, with but two animals recovering. Two stallions and an aged mare and her colt which were confined to the stable and corrals were not affected.

The first symptom noted was the marked odor of onions on the breath of the horses. The respirations were intensified when the animals were forced to move. Constipation or diarrhea was not observed. One of the more severely affected animals showed marked twitching of the shoulder muscles. The urine voided by the affected animals was coffee-colored. The mucous membranes of the mouth and conjunctiva were very anemic and icteric. Temperatures were normal or only slightly elevated. The pulse was full but lacked power.

A citrated (3.8 per cent) blood sample and smears were taken from one young mare which had shown clinical symptoms of poisoning for two days. This mare died a short time later. The erythrocyte count was 2,320,000, and the leukocytes numbered 6,100 per cmm of blood. The hemoglobin by the Tallquist scale was between 30 and 40. The differential count on the basis of 200 cells was as follows: polymorphonuclear leukocytes, 72.5 per cent; lymphocytes, 24 per cent; mononuclear leukocytes, 3.5 per cent. Eosinophils and basophils were absent. The icteric index of the blood-serum was 14.4 as compared with 1.1 and 1.25 for the sera of two apparently normal horses.

A decrease in the erythrocytes and hemoglobin has been observed in the blood of dogs seven to ten days following the feeding of 15 to 20 gm of onions per kilogram of body weight per dog (Sebrell[6]).

The urine collected from a male colt at the time of death was deep reddish-brown. Albumen was present, but no sugar was found.

Autopsies were performed on two horses which had been in a warm rendering plant overnight. Postmortem changes were so far advanced in the internal organs that it was impossible to make a satisfactory pathological examination. The subcutaneous tissues showed marked icterus. The skeletal muscles were of a pinkish salmon color. The ingesta in the stomach of one of the animals were about three-fourths onions and onion tops. The stomach contents of the other animal showed only a few onions and tops.

## SUMMARY

An outbreak of onion poisoning occurred on a farm where nine head of horses had access to unharvested onions. The mortality was high, seven of the nine affected animals dying. The sick animals showed anemia, icterus and coffee-colored urine. These observations confirm the findings of other workers who report losses in cattle and anemia in dogs.

## ACKNOWLEDGMENT

The authors desire to express their thanks to Drs. A. W. Deem and Frank X. Gassner for their interest and support throughout this work.

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# AN UNUSUAL CASE OF FOREIGN BODIES IN THE ALIMENTARY CANAL OF A MULE*

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*Division of Veterinary Medicine, Kansas State College*

This report is being presented with the hope that it will be of sufficient interest to justify its publication. We believe that the presence of foreign bodies in the alimentary canal of members of the equine species is a rare occurrence. This is the first time that the condition has been observed by the author, as well as other members of the staff.

*Subject:* Male mule, aged.

*History:* The mule was purchased, along with a number of other mules and horses, for dissecting purposes. The animal was in fair condition and did not manifest any symptoms of acute digestive disturbance or any other acute derangement while in our care, previous to preparation for dissecting purposes. The owner's reason for disposing of the animal is not known. Neither is it known whether or not this animal ever manifested colic-like symptoms previous to our disposal.

### POSTMORTEM FINDINGS

During the study of the viscera, the foreign objects shown in the accompanying photograph were found. As may be seen, these consisted of old rusty nails, a few pieces of tin, and part of a rusty screw. Figure 1 shows two nails that were found in the pylorus of the stomach. Those shown in figure 2 were found in the ventral part of the great colon, at the sternal flexure, while those in figure 3 were found at the apex of the cecum.

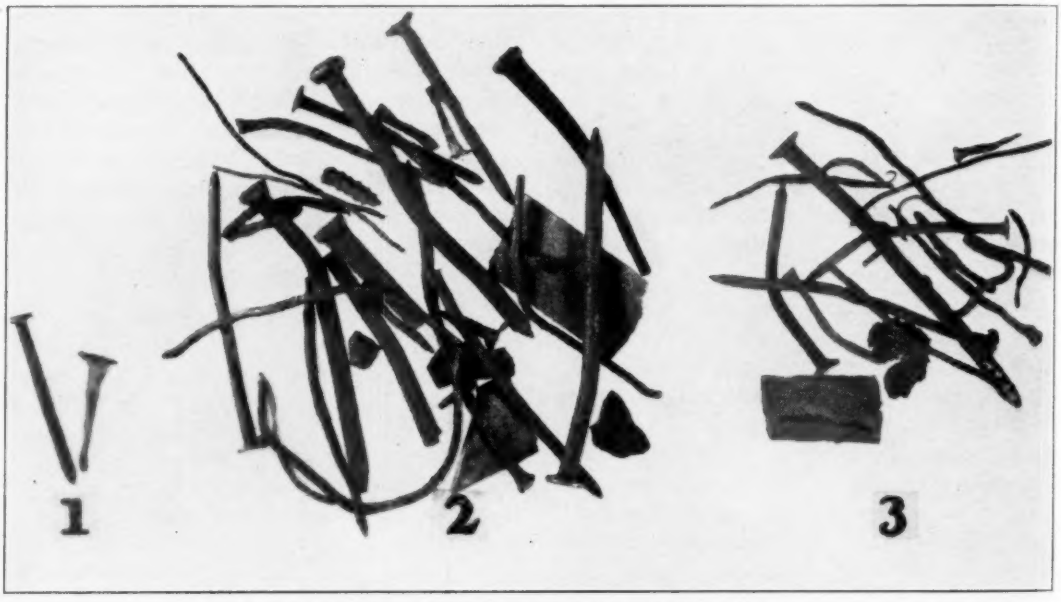


FIG. 1. Two nails found in pylorus of stomach.
FIG. 2. Nails and other objects found in ventral part of colon.
FIG. 3. Foreign bodies found in apex of cecum.

The only lesions present were a chronic inflammation of the pylorus and a fistulous tract through the wall of the cecum, with an adhesion of the base of the cecum to the right ventral part of the great colon. The latter was approximately 2 inches in diameter and was probably produced by one of the objects penetrating the cecal wall at this point, thus producing a localized peritonitis resulting in the adhesion.

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## SEASONAL REMARKS ON HOG CHOLERA AND SWINE INFLUENZA*

*By* R. M. HOFFERD, *Cedar Rapids, Iowa*

This is the season of the year when we should be mindful of swine influenza and especially its possible relation to hog cholera. Swine influenza in itself does not cause heavy death losses if proper care is given the drove, but the loss in body weight is the greatest financial loss. Year after year, however, we encounter cases that no doubt start as swine influenza, but before the disease has run its course hog-cholera complications develop. In such instances losses are indeed very extensive.

In my opinion these cases are grave, considering the loss the owner entails and the damaging effect that may result to the reputation of the veterinarian. Practitioners should observe influenza droves very closely for suspicious symptoms and postmortem lesions of hog cholera, especially if the drove has not been immunized previously against cholera. If the drove has not been immunized, if cholera outbreaks have occurred in the community on nearby farms and if suspicious findings of hog cholera are noted, we should not delay in advising immunization against hog cholera.

The recommended dosage of serum, of course, should be increased 50 to 60 per cent and virus also should be administered. There is practically no risk entailed in treating a drove affected with influenza in this manner. A stimulating effect may be expected from the serum and, by the time the virus reaction starts, the influenza will have run its course.

On the other hand, a drove of swine that develops influenza during the reaction of the serum-virus treatment may be in a "bad way." In these instances losses are apt to be very extensive. At this season of the year, this may occur through no fault of the practitioner, but he can prevent much loss by administering an ample dosage of serum rather than adhering too closely to the label dosage.

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Another thing at this season: Hog cholera is usually more prevalent and, when hogs are running in the cornfields, usually they are not observed so closely as at other times. An owner may wish to have his drove vaccinated because of the close proximity of the disease, and he should be cautioned that his drove may be in the incubation stage of hog cholera. An explanation of this kind probably would be more effective at this time than after losses have occurred following immunization. Also, as stated above, the owner may well be advised that at this season influenza possibly may develop following immunization.

Of course we all admit that pigs should be immunized at an early age, but every year many owners attempt to "get by" without this expense, and in the fall, when the hogs have been turned into the cornfield and hog cholera becomes prevalent, then these owners decide to vaccinate. It is my opinion that it is not advisable to permit hogs to continue to have free access to corn in this manner following the serum-virus treatment.

I recall a case that occurred last fall which may serve to illustrate that a drove affected with influenza may be given the serum-virus treatment safely. One hundred shoats came down suddenly with violent coughing, inappetence, high temperature, and great prostration. In a few days, they exhibited suspicious symptoms of hog cholera, that is, staggering gait, knuckling of the fetlocks and marked depression. On autopsy, suspicious lesions of hog cholera were noted—petechiation of the membranes, organs and lymph-glands. The serum-virus treatment was advised as the safest procedure, with increased dosage of serum. The dosage was increased about 100 per cent and the drove made a good recovery, with a subsequent loss of only three animals.

## SALT POISONING IN HOGS*

*By* R. M. HOFFERD, *Cedar Rapids, Iowa*

*History:* One hundred twenty-five large shoats were in the drove, which had been cholera-immunized after weaning and had

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been doing fine until six days before the writer saw the drove. The owner first noticed several off feed. Two days later, he observed that 15 more were sick. Next day one shoat died. The owner reported that he had purchased a mineral feed and started feeding it five days before the sickness was noted. He said he had put about a quart of the mineral feed in 25 or 30 gallons of slop twice daily. After slopping the drove, he would prepare the slop for the next feeding. The mineral, of course, was always added and the mixture allowed to stand until the next slopping. The mineral feed contined 20 per cent common salt.

*Symptoms:* Most temperatures were normal but some were subnormal. Some had profuse diarrhea. They would lie around, but seemed lively when aroused and were not depressed. Their eyes were bright, but countenance distressed. Some animals were very weak. There was no coughing.

*Postmortem findings:* There was an extensive gastritis in all animals examined. In one shoat the gastric mucosa was extensively eroded and burned in areas, as though some highly irritating substance had been taken into the stomach.

*Recommendations:* The owner was instructed to discontinue the mineral feed and give the drove slop or soaked whole oats for a week. He was told to resume the regular diet gradually and exercise care in the feeding of salt or irritating chemicals.

There were no new cases after the mineral feed was discontinued. Ten animals died and the remaining sick recovered.

## NASAL POLYPUS IN A CHIMPANZEE*

*By* ROY E. NICHOLS, *Columbus, Ohio*
*College of Veterinary Medicine, Ohio State University*

On April 6, 1938, a 45-pound, eleven-year-old, male chimpanzee was presented at the Ohio State University Veterinary Clinic. In the right nostril was a round, fatty-looking growth which nearly occluded the

*Received for publication, November 10, 1938.

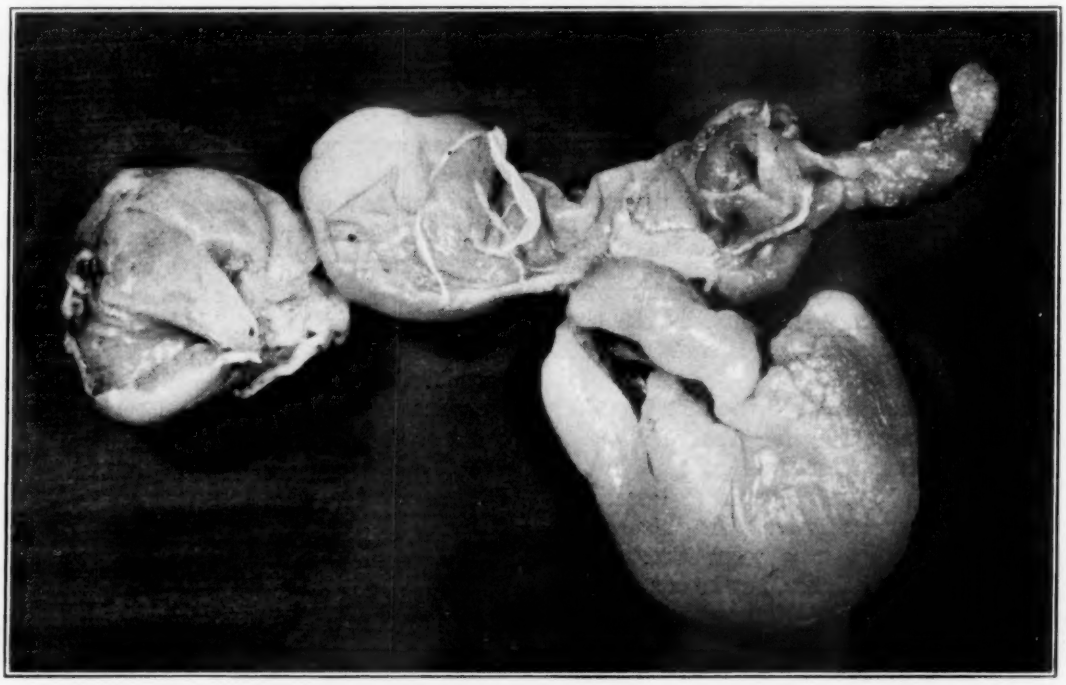


FIG. 1. Nasal polypus removed from a chimpanzee.

nasal opening. Respirations were somewhat difficult. Because the animal was constantly picking at the enlargement, the visible portion was quite red.

Four cc of nembutal was administered into the median vein just below the elbow. This maintained anesthesia for one-half hour. An additional cubic centimeter was administered by the same route as soon as the animal began to struggle. This additional amount was enough to maintain anesthesia until completion of the operation.

The lateral side of the nostril was slit one inch posteriorly to enable better entrance into the nasal passage. Exterior portions of the mass could be palpated with the index finger. The base apparently was attached to the wall of the nasal passage near what corresponds to the malleolar process of the pterygoid portion of the palatine bone. Because of difficulty in reaching the place of origin, forceps were used to strip the base away from the wall. Forceps were used also to remove any rough spicules in that region. Because bleeding was not profuse, the wound was allowed to heal without any attempt to control hemorrhage. The incision in the nostril was sutured with chromic gut and healed very nicely.

The animal apparently was quite conscious within a few hours following the operation and could breathe with much greater ease. Food and water were taken the following day and every day thereafter. The growth, about 4 inches in total length, was of the consistency of fat. At the base was an area containing what appeared to be and what felt like fibrous tissue and cartilage. Upon being sectioned, the fatty-like tissue was classified as fibromyxoma and the harder tissue as cartilage.

## COW ADOPTS FAWN*

*By* L. J. TOMPKINS, *Packanack, Lake, N. J.*

During a recent trip to Michigan, a very interesting and unusual case of an adoption was brought to my attention. A cow on the farm of Ivan Snyder, near Marion, Mich., had adopted a fawn. As far as could be learned, this had been a purely voluntary act upon the part of the cow. The adoption took place in the pasture.

When first observed by the owner of the cow, the fawn did not appear to be much over a month old. It became quite tame and showed no fear of persons. A later report states that the fawn weighs about 100 pounds and is still nursing the cow. It is believed to have grown much more rapidly as a result of nursing the cow than would have been the case if it had been nursed by its own mother. The fawn is a buck and has stubs of horns an inch long. The foster mother is very fond of it.

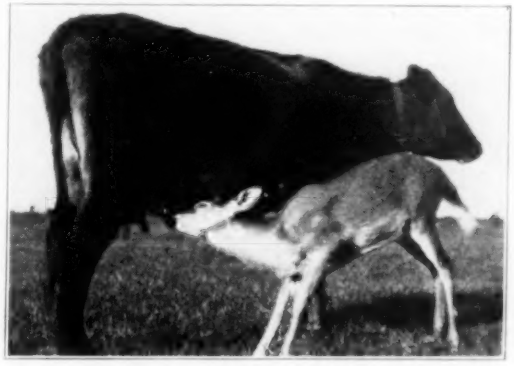


FIG. 1. Fawn nursing foster mother.

The accompanying photograph was taken by Van de Warker, of Marion, Mich.

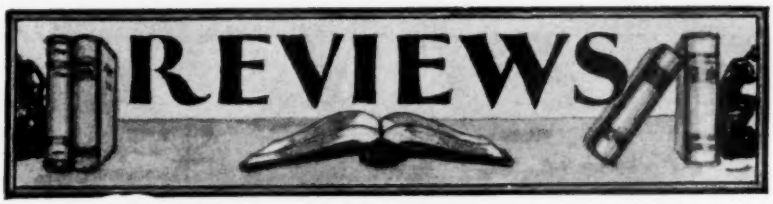
*Received for publication, November 21, 1938.

## A PECULIAR ACCIDENT*

*By* CHAS. HAASJES, *Shelby, Mich.*

Recently, a farmer called me and stated that one of his horses had a nail in its eye. Upon arrival at the farm, I found a ten-year-old gelding with a ten-penny nail through the lower eyelid. The horse appeared normal but had broken the manger during the previous night. The remarkable part of it was that the nail had pierced the eyelid from the inside about one and one-half inches from the edge of the lid, without injuring the eyeball. The nail was easily removed after an incision was made in the lid to enlarge the opening. No serious results followed the accident.

*Received for publication, September 1, 1937.



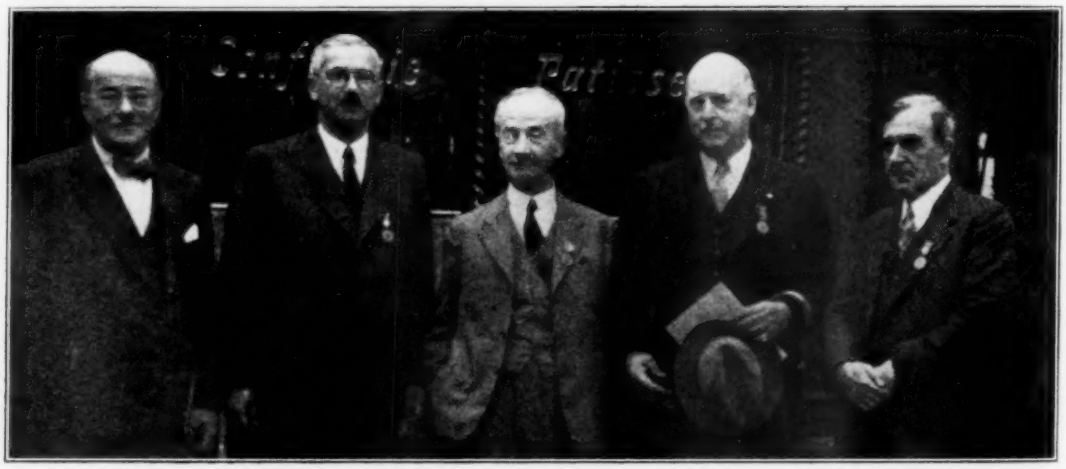

PATHOLOGY AND THERAPEUTICS OF THE DISEASES OF DOMESTIC ANIMALS. Dr. Franz Hutyra, Ancient Royal Veterinary College, Budapest; Dr. Joseph Marek, Royal Hungarian Palatin-Joseph University, Budapest; and Dr. Rudolph Manninger, Royal Hungarian Palatin-Joseph University, Budapest. 4th English edition, 3 volumes. 2,519 pages, illustrated. Alexander Eger, Chicago, 1938. Cloth, $31.50.

Since the volumes known as "Hutyra and Marek" have occupied a place of preëminence for many years throughout the world where veterinary medicine is read and studied, a brief review of this "encyclopedia of veterinary science" can deal only with generalities. The wealth of details which have placed its name among the classics of this period of veterinary-medical history one leaves to its well-established reputation.

A glance at the prominent figures connected with the authorship underwrites the authenticity of its text, and the tables of contents of the volumes, containing, respectively, 962, 704 and 761 pages covering, without notable exception, all diseases of domestic animals throughout the civilized world, insure its completeness.

The addition of Professor Manninger to the authors has brought new thoughts, new diction and new material to the context of many a subject, and the editing of Professor Greig and his American coworkers, Mohler and Eichhorn, shows outstanding superiority over previous editions. One looks in vain for the literary infelicities which so often creep into vast translations of technical material.

In assuming the responsibility of reviewing this welcome contribution to the literature of our profession, the writer finds that the best indorsement is revealed in the lives of the men who have labored so faithfully to give us this work on veterinary medicine, and in reading critically the text of subjects with which one is most familiar,



Left to right: DR. ADOLPH EICHHORN, EDITOR; PROF. DR. RUDOLPH MANNINGER, CO-AUTHOR; DR. JOSEPH MAREK, CO-AUTHOR; DR. J. R. MOHLER, EDITOR; DR. J. RUSSELL GREIG, EDITOR-IN-CHIEF, FOURTH ENGLISH EDITION, "SPECIAL PATHOLOGY AND THERAPEUTICS OF THE DISEASES OF DOMESTIC ANIMALS." (Photograph taken at Interlaken, Switzerland, during the Thirteenth International Veterinary Congress, by Dr. D. M. Campbell.)

one is indeed struck with the authors' historical and scientific precision, not to mention their fairness in allocating credits.

To say the least, "Hutyra, Marek and Manninger," as these volumes will no doubt be known in the future, is an extraordinary piece of work, without counterpart, which should find a place in the library of everyone at all interested in animal pathology. As a matter of fact, just how a capable veterinarian can deny himself the benefit of having these volumes within reach is inconceivable.

Volume I, largest of the three, describes the infectious diseases which the authors divide into: (1) Acute General Infections; (2) Acute Exanthematous Infections; (3) Infectious Organic Diseases; (4) Infectious Nervous Diseases; (5) Chronic Infections; and (6) Protozoal Diseases.

Volume II covers diseases of the digestive tract, including helminthiasis, in five sections; of the accessory organs of digestion, in three sections; diseases of the respiratory tract, in five sections; and of the heart and blood-vessels, in three sections.

Volume III is essentially a treatise on organic diseases: of the kidneys; the bladder; the blood and blood-producing organs; the spleen; the endocrine glands and metabolism; the nervous system, in four sections; organs of locomotion; and skin. While none of the chapters is lacking in any important respect, the 230 pages on diseases of the skin which terminate volume III will be found interesting, up-to-date reading and, as one would expect of the editor-in-chief, the chapter on milk fever has been revised freely to conform to the popular conception of that nervous upset of the dairy cow. Throughout, one is fascinated with the personal opinions of great authors in the field of animal pathology and therapeutics.

In regard to the title of this well-known *chef d'ouvre* of veterinary literature, the writer continues to wonder if the phrase "of the diseases" is not a pleonasm.

L. A. M.

## PUBLICATIONS RECEIVED

Studies on Fowl Paralysis. 3. A Condition Resembling Osteopetrosis (Marble Bone) in the Common Fowl. Erwin Jungherr and Walter Landauer. (Tech. Bul. 222. Storrs Agr. Exp. Sta., Feb., 1938. pp. 34. Illus.)

Infection of the Chicken with *Capillaria Columbae* (Rud.). P. P. Levine. Reprint from *Jour Parasitol.*, xxiv (1938), 1, pp. 45-52.

Calcium Borogluconate. James Stewart and Herbert Taylor MacPherson. Reprint from *Vet. Jour.*, xciv (1938), 6, pp. 220-222.

On the Nutrition of the Fowl Nematode *Ascaridia Lineata* (Schneider). James E. Ackert. Reprint from *Trans. Amer. Micro. Soc.*, lvii (1938), 2, pp. 218-222.

Light as a Factor in the Production of Pigment by Certain Bacteria. James A. Baker. Reprint from *Jour. Bact.*, xxxv (1938), 6, pp. 625-631.

Some Notes on Sarcosporidia in Virginia. I. D. Wilson and Ruth McDonald. Reprint from *Jour. Parasitol.*, xxiv (1938), 3, pp. 249-250.

The Vitamin A Content of the Colostrum of Dairy Cows. James Stewart and Jennie Whitelaw McCallum. Reprint from *Jour. Agr. Sci. (Eng.)*, xxviii (1938), 3, pp. 428-436. Illus.

Growing Larval *Ascaridia Lineata* (Nematoda) *in Vitro*. J. E. Ackert, A. C. Todd and W. A. Tanner. Reprint from *Trans. Amer. Micro. Soc.*, lvii (1938), 3 pp. 292-296.

The Classification of Acid-Fast Bacteria. Ruth E. Gordon and William A. Hagan. Reprint from *Jour. Bact.*, xxxvi (1938), 1, pp. 39-46.

Quantitative Studies of Brucella Precipitin Systems. I. Precipitation of Homologous Antisera by Brucella Endoantigens. R. B. Pennell and I. F. Huddleson. Reprint from *Jour. Exp. Med.*, lxviii (1938), 1, pp. 73-81. Illus.

Quantitative Studies of Brucella Precipitin Systems. II. The Precipitation of Heterologous Antisera by Brucella Endoantigens. R. B. Pennell and I. F. Huddleson. Reprint from *Jour. Exp. Med.*, lxviii (1938), 1, pp. 83-93. Illus.

Dysentery of New-Born Lambs. (Circ. 153. Mont. State Col. Agr. Exp. Sta., July, 1938. pp. 8. Illus.)

Dysentery of New-Born Lambs. H. Marsh and E. A. Tunnicliff. (Bul 361. Mont. State Col. Agr. Exp. Sta., July, 1938. pp. 42.)

An Improved Medium for the Storage of *Actinomyces Necrophorus* Cultures. E. A. Tunnicliff. Reprint from *Jour. Inf. Dis.*, lxiii (1938), pp. 113-116.

Stallion Enrollment and the Horse-Breeding Situation. S. R. Speelman. (U. S. Dept. Agr., Washington, D. C., 1938. pp. 24.)

Some Observations on Manson's Eyeworm of Poultry in Antigua, B. W. I., and a Suggested Method of Control. L. R. Hutson. Reprint from *Trop. Agr.*, xv (1938), 3, pp. 66-68.


AN ENDOTOXIN FROM ASPERGILLUS FUMIGATUS. Arthur Henrici. Jour. Bact., xxxvi (1938), p. 278.

Cell sap from a pathogenic strain of *Aspergillus fumigatus* was toxic for rabbits, guinea pigs, mice and chickens, while the broth filtrate is harmless. Pathogenic strains of *Aspergillus flavus* and *Aspergillus oryzae* showed a similar but lesser toxicity. The cell sap is hemolytic *in vitro*. Injected subcutaneously in rabbits it produces a massive gelatinous exudate similar to that resulting from *Clostridium oedematiens* toxin with central purpura and necrosis. Intravenously and intraperitoneally large doses cause death within 48 hours with pulmonary congestion and hemorrhage. In case of smaller doses death is delayed up to two weeks and fatty changes with necrosis are found in liver and kidneys. An extensive serofibrinous exudate in the pleural and peritoneal cavities is common but not constant. Guinea pigs often develop paralysis which becomes general before death. The toxin is neutralized by sodium ricinoleate. The toxin closely resembles that of *Amanita phalloides*. It differs, however, in being heat labile and non-toxic by mouth. It resists 55° C. for 45 minutes but is inactivated at 62° C. for 15 minutes. Rabbits and guinea pigs may be immunized actively so that they tolerate ten or more lethal doses.

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PRODUCTION OF ANTISERUM FOR EQUINE ENCEPHALOMYELITIS. Edward Records and L. R. Vawter. Jour. Bact., xxxvi (1938), p. 295.

Antisera of high neutralizing titre will probably be needed, for some time to come, as an aid in the treatment of clinical cases of equine encephalomyelitis. Even after a prolonged course of immunization by the subcutaneous injection of virus into horses, the response is poor or variable and the sera of only a few animals attain a titre considered satisfactory for clinical use. It has been observed that a single intravenous injection of a large dose of equine encephalomyelitis virus into immune horses results in a ten-fold rise of antiviral titre in ten days. No fatalities or unduly severe reactions in the injected horses occurred. Titration of the virus-neutralizing power of individual and pooled sera from hyperimmune horses was conducted on both ten-day-old chick embryos and guinea pigs. The chick embryo is considered equally as dependable as the guinea pig, if not more so. The use of embryos is more economical and rapid.

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THE EFFECT OF THE SEX HORMONES ON THE RENAL EXCRETION OF ELECTROLYTES. George W. Thorn and Lewis L. Engel. Jour. Exp. Med., lxviii (1938), p. 299.

In normal male dogs subcutaneous injections of progesterone, estrone, estradiol or testosterone proprionate were followed by a decreased renal excretion of sodium and chlorides. The compounds differed markedly in their potency and in the deviation of the effect following a single subcutaneous injection. The injection of estrone, estradiol or testosterone proprionate was followed by a decreased renal excretion of inorganic phosphorus and total nitrogen. On the day of injection, a slight increase of renal excretion of potassium frequently followed administration of progesterone, estrone, estradiol or testosterone proprionate. Experiments on suprarenalectomized dogs indicated that the effect of the sex hormones on the renal excretion of electrolytes was not necessarily mediated through the suprarenal gland. With the possible exception of progesterone, none of the compounds

studied was effective in prolonging the life of suprarenalectomized male dogs.

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ALTERED CUTANEOUS CONDITIONS IN THE SKIN OF TUBERCULOUS GUINEA PIGS AS DEMONSTRATED WITH A VITAL DYE. A. L. Joyner and F. R. Sabin. Jour. Exp. Med., lxviii (1938), p. 325.

The spread of a vital dye, pontamine sky blue, and the drainage of the dye into the vascular system takes place much more slowly in the skin of the tuberculous guinea pig while it is allergic than in a normal animal. In the skin of moribund tuberculous guinea pigs, animals no longer allergic, the dye spreads more rapidly than in the normal animal. The spread of the dye was somewhat restricted in the skin of guinea pigs infected with a hemolytic streptococcus. The animals were allergic. The findings suggest that the dye method may disclose altered tissue conditions in the allergic state.

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THE EFFECT OF THE PULSE ON THE FORMATION AND FLOW OF LYMPH. Robert J. Parsons and Philip D. McMaster. Jour. Exp. Med., lxviii (1938), p. 353.

The ears of rabbits were perfused with defibrinated rabbit blood in such a way that pulsation could be imparted to the perfusate or withheld from it at will. In the absence of pulsation there was almost no lymph flow, whereas when it was present lymph flow was rapid despite the fact that the "systolic" pressure of the perfusate never exceeded the constant pressure in the non-pulsatile instances and the volume flow was far less. Non-pulsatile perfusion led to a slight flow of lymph in ears that were becoming edematous, whereas when it was pulsatile the lymph flow was enormous. The pulse exercises an influence to move fluid into the lymphatics and also along them.

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THE EFFECT OF THE PULSE ON THE SPREAD OF SUBSTANCES THROUGH TISSUES. Philip D. McMaster and Robert J. Parsons. Jour. Exp. Med., lxviii (1938), p. 377.

The pulsation of blood-vessels in the ear of the rabbit greatly increased the rate of the spread of dye introduced into the subcutaneous tissues. The appearance of edema in tissues perfused at a constant pressure led to very little increase in the rate of the dye spread. By contrast, a rapid interstitial spread of dye occurred in tissues becoming edematous while perfused with a pulsatile flow of blood.

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THE BEHAVIOR OF POX VIRUSES IN THE RESPIRATORY TRACT. 1. The response of mice to the nasal instillation of vaccinia virus. John B. Nelson. Jour. Exp. Med., lxviii (1938), p. 401.

A catarrhal reaction manifested by a coryza and a pneumonia of characteristic pathology was regularly produced in mice by the nasal instillation of vaccinia virus. Inoculation into embryonated eggs indicated that the virus entered the circulation as early as the second day after injection. The vaccinial catarrh was readily transmissible by the passage of nasal exudate but not by contact. Dosage was important in establishing the virus in the nasal passages, the limiting dilution being approximately $10^{-3}$ of an egg-membrane suspension (at least 1,000 times the amount required to infect an embryonated egg). The morbidity rate was variable but in general high, reaching 70 per cent in two groups of 50 mice. An immunity which was effective against reinfection for several months but ultimately declined was attendant on recovery. The amount of virus required to produce this immunity was significantly less than the infective dosage.

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BLOOD PLASMA PROTEINS AS INFLUENCED BY LIVER INJURY INDUCED BY CARBON TETRACHLORIDE AND GUM ACACIA. C. C. Erickson, G. P. Heckel and R. E. Knutti. Amer. Jour. Path., xiv (1938), p. 537.

Frequent oral administration of carbon tetrachloride to dogs produced moderate cirrhotic changes in the liver. In such animals the plasma protein concentration fell slightly and this decrease appeared to be due largely to the loss of albumin. The continued administration of gum acacia combined with carbon techrachloride re-

sulted in deposition of what appeared to be acacia in liver cells as in the case of the administration of gum acacia alone. Other sites of acacia deposits in dogs receiving carbon tetrachloride and acacia appeared to be in the sinusoidal lining cells of the spleen and in the large mononuclear phagocytic cells which were found in sparse numbers in the lungs, spleen, lymph-nodes and bone-marrow. There was no evidence of edema although bleeding time was prolonged. The reactions of albumin, globulin and fibrinogen associated with various types of liver injury indicated that those substances may be produced independently of each other and that the liver is concerned in their production.

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THE SUSCEPTIBILITY OF VOLES TO HUMAN AND BOVINE STRAINS OF TUBERCLE BACILLI. A. Q. Wells. Brit. Jour. Exp. Path., xix (1938), p. 324.

The amount of human tubercle bacilli which will produce demonstrable disease after intraperitoneal injection in a vole after one month is at least one milligram. Extensive and progressive disease resulted one month after injection in all voles which were injected with bovine tubercle bacilli in amounts varying from 1 mg to 0.00001 mg. The author suggests a dose of 0.001 mg moist weight of culture of tubercle bacilli, injected intraperitoneally into voles, will distinguish unfailingly between human and bovine tubercle bacilli in one month.

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THE PATHOLOGY OF EASTERN EQUINE ENCEPHALOMYELITIS IN THE GUINEA PIG. L. S. King. Amer. Jour. Path., xiv (1938), p. 636.

Special attention was paid to the pathologic findings of the central nervous system in the early stages of the disease before symptoms were apparent. After subcutaneous inoculation of the virus, the typical lesion is an isolated, fairly well circumscribed focus of polymorphonuclear leukocytes found principally in the cerebral cortex, although any portion of the brain may be affected. In such foci the damage to neurones appears to be secondary to the inflammatory reaction. Two types of lesions are clearly distinguishable, the inflammatory and the degenerative, although there may be a certain degree of overlapping. The inflammatory response is considered to be the primary reaction to the disease agent after peripheral inoculation. Analysis of the distribution of lesions in early, symptomless cases shows that the virus passes directly from the blood into the brain tissue. The presence of lesions in the cerebral cortex with intact subcortical centers is held to exclude nerve transmission. Under certain conditions the virus may travel along nerve paths.

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RATE OF ABSORPTION OF CARCINOGENS AND LOCAL TISSUE REACTIONS AS FACTORS INFLUENCING CARCINOGENESIS. P. R. Peacock and Stephan Beck. Brit. Jour. Exp. Path., xix (1938), p. 315.

The induction of sarcoma in the connective tissues of mice following the injection of 3:4 benzpyrene in various solvents depends more upon the rate of absorption of the benzpyrene than upon the early tissue reaction to the solvent. When benzpyrene is eliminated within three months, tumors are rare (five out of 41 surviving for four months); when elimination is delayed beyond six months, tumors are common (34 out of 46 surviving for four months). Tumors occur at a point of optimum concentration of benzpyrene which in these experiments was found to be a few millimeters away from the principal focus of benzpyrene.

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ON THE FAILURE OF ACUTE AND SUBACUTE INFLAMMATION TO INFLUENCE CARCINOGENESIS WITH 3:4 BENZPYRENE. Stephan Beck. Brit. Jour. Exp. Path., xix (1938), p. 319.

Mice were injected with 3:4 benzpyrene dissolved in a 30 per cent solution of turpentine in olive oil, benzpyrene near or into an abscess, and benzpyrene without turpentine. Controls received no benzpyrene. All groups injected with benzpyrene developed tumors and showed the fluorescence of per-

sistent benzpyrene in the tissues for about six months. Acute inflammation apparently has no influence on the rate of absorption and on the carcinogenicity of the benzpyrene in fatty solvents. The inflammatory fibrous tissue did not accelerate or retard the development of sarcoma. Sarcoma did not originate from the fibrous tissue of subacute inflammation. Benzpyrene did not act as the locus of injection but on the tissues in the immediate neighborhood through which it passed on its way to absorption. The agent titrates itself against the tissues and an optimal concentration for an optimal time determines which of the susceptible cells become malignant.

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DEGENERATIVE ARTHRITIS. A comparison of the pathological changes in man and equines. George R. Callender and R. A. Kelser. Amer. Jour. Path., xiv (1938), p. 680.

The changes of degenerative arthritis found in 60 knee joints from man and in various joints from 54 horses and mules were studied. The lesions were of practically identical types in man and equines, though advanced degeneration was seen only in man, as the equines developed such disability that they were destroyed before such advanced changes occurred. Hypertrophic changes were considered to be secondary to loss of substance and consequent malocclusion of the joint and are believed to be of a compensatory effort to maintain the proper distribution of the joint stress. This hypertrophy consisted of bone growth replacing destroyed cartilage and extending joint surfaces. It appeared to start in the calcified matrix and to extend into overlying degenerated cartilage. Considerable joint change was often found in both man and animals without history of symptoms. The incidence of degenerative arthritis in man agreed with that of Keefer and Parker. None was found in persons under 20 years of age; there was rapid increase up to the age of 40, and thereafter some change was found in practically every knee joint examined. Only one horse below the age of ten years was examined and that one was the only one without joint lesions.

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ANEMIA STUDIES WITH DOGS. V. R. Potter, C. A. Elvehjem and E. B. Hart. Jour. Biol. Chem., cxxvi (1938), p. 155.

After growing dogs had developed anemia, after being fed on milk diets with rigid exclusion of iron and copper, they were placed on various combinations of iron and copper, the highest of which was 30 mg of iron and 2 mg of copper per day. The rate of hemoglobin regeneration over a period of three months was such that it was felt that either the levels of iron and copper were too low or that a third or unknown factor was lacking.

After the hemoglobin levels had approached normal, therapy was discontinued and two of the dogs were made anemic by bleeding, while the remainder of the animals were maintained on whole milk alone. The anemic dogs apparently were well depleted of both iron and copper and were virtually unable to form hemoglobin on the milk diet alone. The dogs which were not bled appeared able to maintain their hemoglobin levels on an extremely low intake of iron and copper on the milk alone. The dogs which were made anemic by bleeding responded poorly to a daily dose of 30 mg of iron alone but, when 4 mg of copper per day was given in addition, the hemoglobin regeneration proceeded very rapidly.

The canine genus should be included among those which require copper for hemoglobin regeneration and whose need for copper for hemoglobin synthesis is probably a general biological property. Blood copper determinations showed that an increase in blood copper is associated with accelerated hematopoiesis. Dogs were maintained for over a year in a state of excellent nutrition on a diet of whole milk with the additions of vitamins A and D, with iron or copper, or both, fed during periods of therapy.

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Moderation is the silken string running through the pearl-chain of all virtues.—*Fuller.*


## Regular Army

Captain John H. Rust, III, is relieved from his present assignment and duty at Fort Slocum, N. Y., effective at such time as will enable him to proceed to New York, N. Y., and sail on or about February 7, 1939, for the Hawaiian Department, and upon arrival to report to the commanding general for assignment to duty with the Veterinary Corps.

Captain Edgerton L. Watson is assigned to duty at Fort Slocum, N. Y., effective upon completion of his present tour of foreign service in the Hawaiian Department.

The appointment of 1st Lieut. Karl Harry Willers, Veterinary Corps Reserve, as first lieutenant in the Veterinary Corps, Regular Army, with rank from November 1, 1938, and his assignment to station at Fort Oglethorpe, Ga., are announced. Lt. Willers, now on duty at Van couver Barracks, Wash., will proceed at the proper time to San Francisco, Calif., and sail on the transport scheduled to leave that port on or about November 29, 1938, for New York, N. Y., and upon arrival will proceed to Fort Oglethorpe, and report for duty.

By direction of the President, Colonel Alfred L. Mason, V. C., upon his own application is retired from active service, to take effect November 30, 1938, under the provisions of section 1243, Revised Statutes, after more than thirty years' service. He is relieved from his present assignment and duty at the Presidio of San Francisco, Calif., on November 30, 1938, and at the proper time will proceed to his home.

1st Lieut. Benjamin F. Leach is relieved from assignment and duty at Fort Sheridan, Ill., and from temporary duty at Fort Knox, Ky., effective in time to comply with this order, is then assigned to the Medical Field Service School, Carlisle Barracks, Pa., and will proceed to that station and report on January 13, 1939, to the commandant, Medical Field Service School, for the purpose of pursuing the basic course of instruction.

The promotion of Major Nathan M. Neate, to the grade of lieutenant colonel, with rank from November 24, 1938, is announced.

The following veterinary officers of the National Guard have been enrolled in the Forage Inspection Course at the Army Veterinary School, Army Medical Center, Washington, D. C., beginning November 28, 1938:

Captain Truman I. Means, 111th Cav., New Mexico N. G.

Captain Geo. S. Mechling, Vet. Co., 112th Med. Regt., Ohio N. G.

Captain Chas. H. Kitselman, Med. Dept. Det., 114th Cav., Kansas N. G.

The following named officers of the Veterinary Corps are directed to proceed at the proper time from Washington, D. C., to Carlisle Barracks, Pa., and report on January 13, 1939, to the Commandant, Medical Field Service School, for temporary duty for a period of approximately five months for the purpose of pursuing the basic course of instruction:

Captain Thomas C. Jones

Captain Donald C. Kelley

Captain Wayne D. Shipley

1st Lieut. Walter T. Carll

## Veterinary Corps Reserve

NEW ACCEPTANCES

(First lieutenants)

Boston, Lawrence Duke, Bureau of Animal Industry, Federal Building, Oklahoma City, Okla.

Durrant, Marvin John, Dows, Iowa.

Fitch, James Adams, 2111 Knapp St., Saint Paul, Minn.

Hinson, Conrad Raymond, 212 Hendrix St., Millen, Ga.

Merritt, Walter Edward, 1130 S. 19th St., Fort Dodge, Iowa.

Robinson, Virgil Benton, RFD No. 5, Clanton, Ala.

Turner, Joe Gordon, 2121 Cedar Springs, Dallas, Texas.

SEPARATIONS

Major Eugene Leo Hannon, died October 2, 1938. Captain Walter Emmert Neary, died November 6, 1938.

PROMOTIONS

*To Captain:* Morris Elmer Blostein, 819 S. Main St., Horseheads, N. Y.

*To Captain:* Robert Milton Parker, 509 S. Myrtle St., Columbia, S. C.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

1st Lieut. Edgar Wm. Millenbruck, Fort Leavenworth, Kan.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

1st Lieut. Willard H. Merchant, Camp Custer, Mich. 1st Lieut. Karl H. Willers, Vancouver, Wash. (Accepted appointment in Regular Army.)


## VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The October meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, on Wednesday evening, October 5, 1938.

President Robert S. MacKellar introduced the guest speaker, Dr. M. A. Emmerson, Assistant Professor of Veterinary Surgery and Obstetrics at the University of Pennsylvania, who talked on "The Required Technic and Necessary Equipment in X-Ray Therapy of Domestic Animals." He gave a cineographic demonstration of the method and procedure in x-ray therapy as applied to both large and small animals. Dr. Emmerson then gave specific references to four cases:

The first case was a cow suffering from actinomycosis of the maxillary sinus, treated with a total of 5,237 r (Roentgen unit) given in five treatments, over a period of approximately one month.

The second case was a cow suffering from actinomycosis of the right mandible. She received a total of 6,331 r in four treatments, in a period of three weeks.

The third was a four-year-old Scottie suffering from cancer of the skin of the paw. After receiving a total of 812 r in two treatments, in a period of one week, the dog was cured. Now, according to Dr. Emmerson, no one would be able to detect where the carcinoma had existed.

Case 4 was an eleven-year-old collie, suffering from an extensive skin cancer in the region of the paw. After receiving a total of 2,526 r, in two weeks, the dog recovered and there have been no signs of a recurrence. Dr. Emmerson pointed out the fact that, in the cases of the dogs, new hair of apparently the original color had formed over the affected regions, whereas in the bovine cases, the new hair was white instead of the original color.

The members received Dr. Emmerson's talk with enthusiasm and appreciation, because it contained much valuable information on a relatively new field of x-ray therapy.

### November Meeting

The November meeting was held at the Hotel New Yorker, Wednesday evening, November 9, 1938.

Dr. J. W. Landsberg, of the Warner Institute for Therapeutic Research, New York City, was the guest speaker. He presented a paper on "*Ancylostoma Caninum*." He pointed out that the research work and scientific study of hookworm in dogs has been utilized extensively in the solution of problems pertaining to human hookworm disease. Although these are two different parasites, he explained, their life cycles and pathological changes affecting their hosts are almost identical.

The speaker stated that hookworm larvae are found in the upper half-inch of soil and that they attack the host in one of three ways: by penetration of the skin, by the mouth or by prenatal infection. He stated further that the pathology associated with infestation by this parasite is essentially an anemia, brought about by the blood-sucking activity of the worm. The anemia is of two types, either acute or chronic posthemorrhagic, depending upon the magnitude of the infestation.

Dr. Landsberg explained that leukocytosis and eosinophilia may accompany each infestation, although not necessarily, and no change in the thrombocytes has been observed. The anthelmintics recommended are tetrachlorethylene or hexylresorcinol.

That the members sincerely appreciated Dr. Landsberg's paper was shown by the rising vote of thanks they tendered him.

J. B. Engle, *Secretary.*

## WASHINGTON STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Washington State Veterinary Medical Association was held at Yakima, on October 15, 1938. While the primary purpose of the meeting was to administer the business and formulate the policies of the Association, brief reports on current items of interest also were presented.

An election of officers took place with the following results: President, Dr. Russell R. Isham, of Auburn; vice-president, Dr. J. P. Johnson, of Seattle; and secretary-treasurer, Dr. V. C. Pauhlman, of Chehalis.

One of the interesting facts brought out during the meeting was that increased interest in the local as well as the state association is being shown, as evidenced by added membership and improved finances.

Dr. J. C. Exline, inspector-in-charge, U. S. Bureau of Animal Industry, reported on the Bang's disease control program inaugurated on August 16, 1934. Dr. M. R. Hales, Supervisor of Dairy and Live Stock, led a discussion on sporadic cases of equine encephalomyelitis in the state. Dr. H. A. Trippeer, city veterinarian of Walla Walla, reported on a survey of the milk-shed of metropolitan Chicago.

A highlight of the business meeting was the acceptance of 37 applications for membership, thereby exceeding the quota set for the year. Dr. H. W. Marsden, chairman of the Practice Act Commitee, gave a report of progress and announced authorization to proceed with the drafting and presentation of a new act to the State Legislature in 1939. A special fund of $500.00 was partially subscribed to finance the work of this committee.

In the annual report of the Resolutions Committee, the following factors were stressed: (1) Continued promotion of veterinary education facilities at Washington State College; (2) endorsement of state and federal disease control programs involving indemnity payment for cattle reacting to tests for Bang's disease and tuberculosis; (3) promotion of continued progress in the state meat-inspection service; (4) recognition of the establishment at Washington State College of a research project on fur-bearing animals by the U. S. Bureau of Biological Survey, and (5) statement of necessity for coöperation between various health officials in the control of rabies.

A slogan, "Improvement in the Services to the Live Stock Industry," was set forth as the most direct approach to veterinary problems.

V. C. PAUHLMAN, *Secretary.*

## FLORIDA STATE VETERINARY MEDICAL ASSOCIATION

The ninth annual meeting of the Florida State Veterinary Medical Association was held at the El Comodoro Hotel, Miami, October 24-25, 1938.

Hon. Robert R. Williams, mayor of Miami, delivered the address of welcome. The response to Mayor Williams' address was given by Dr. H. C. Nichols, of Ocala. President C. A. Palmer, of Gainesville, then addressed the group.

Dr. J. L. Ruble, of Orlando, spoke on "Diseases of the Prostate Gland and Their Relation to Perineal Hernias." Mr. W. F. Ankerbrand, of the Upjohn Co., Miami, was the next contributor; his subject was "Vitamins." Good discussions followed both speakers.

In the afternoon, a small-animal clinic was held at the Pet Animal Hospital of Dr. A. T. Knowles. This portion of the program, virtually everyone agreed, was the most interesting feature of the entire meeting. Dr. E. D. Clawson, of Miami, demonstrated a complete examination of the dog, including diagnosis. Next, an enterotomy was performed by Drs. C. E. Bild and L. L. Kelley, of Miami.

Inserting a tube into the stomach for the removal of a foreign body and inserting a tube into the cecum for the treatment of whipworm infestation were demonstrated by Drs. W. E. Lord and F. E. Monroe, of Miami.

Other contributors to the clinic were: Drs. S. V. Ramsey, of Miami—cataract operation; W. E. Lord and F. E. Monroe—

hysterectomy, and A. G. Brown, a physician of Miami—several operations on the eye.

In the evening, a banquet and dance was held in the patio of the Legion Home, on Biscayne Bay. Dr. E. D. Clawson officiated as toastmaster.

The program of the second day was opened with a clinic. The contributors were: Dr. L. E. Swanson, head of the Zoölogical Laboratory, Moultrie, Ga.—liver flukes; and Dr. D. A. Sanders, of Gainesville—enzoötic bronchopneumonia in dairy calves. Following the clinic, Dr. I. S. McAdory, of Alabama Polytechnic Institute, Auburn, Ala., addressed the group. "Observations on an Ictero-Hemoglobinuria-like Disease in Florida," by Drs. M. W. Emmel and D. A. Sanders, delivered by Dr. Emmel, was the final presentation at the morning session.

A question-box was a feature of the afternoon session, followed by a business meeting. Honorary membership was conferred upon Drs. A. W. Ziebold, of Miami, and I. S. McAdory.

Upon recommendation of the Legislative Committee, the Association adopted proposed amendments to the Florida Veterinary Practice Act, to be introduced at the session of the State Legislature which convenes in April, 1939.

Sixty-three veterinarians attended the meeting.

J. V. KNAPP, *Secretary.*

## TRI-STATE VETERINARY MEDICAL ASSOCIATION

A meeting of the Tri-State Veterinary Medical Association was held at the Hotel Winona, Winona, Minn., November 17, 1938.

After President George Failing called the meeting to order, Drs. E. C. Rosenow and C. F. Schlotthauer, of the Mayo Clinic, Rochester, Minn., presented a paper on "Equine Encephalomyelitis, Its Cause, Prevention and Its Relationship to Encephalomyelitis as It Occurs in the Human." The discussion was conducted by Drs. C. E. Cotton and H. C. H. Kernkamp, of Saint Paul, Minn.

Professor R. Peterson, of University Farm, Saint Paul, Minn., gave an interesting talk on "The Relationship of Minerals in the Diet of Live Stock," and a very lengthy discussion followed.

In the evening, dinner was served to 71 veterinarians and their wives.

P. H. RIEDE, *Secretary.*

## KANSAS CITY VETERINARY MEDICAL ASSOCIATION

Eighty members of the Kansas City Veterinary Medical Association and their families were guests of the Army Veterinary Corps at Fort Leavenworth, Kansas, on November 19, 1938.

Lt. Colonel George C. Rife and Major V. C. Hill acted as hosts. Colonel D. W. Harmon, of the Medical Corps, extended the welcome in behalf of the officers of the Post, and the response was made by Dr. J. C. Flynn, President. An interesting program had been arranged, consisting of an exhibition of army mounts of the hunter and polo types and an inspection of the hospital and stable management.

The ladies were entertained with a bridge party at the Officers' Club, Mrs. George C. Rife and Mrs. V. C. Hill serving as hostesses. Later in the afternoon, the visitors attended the museum to view many rigs used in the days of the pony express, including a surrey in which Abraham Lincoln rode from Fort Leavenworth to Fort Hays, Kansas.

An outstanding feature of the entertainment was a horse show of class riding and jumping. Lt. Colonel Rife's horse, "Star Dust," was the undisputed champion of the open jump, winning the cup and blue ribbon. Ribbons and cups were awarded in each of the classes. The dressage exhibition of Captain I. L. Kitts, of the Field Artillery, riding "American Lady," was one of the finest numbers of the entertainment. Captain Kitts' mare is considered the best in the United States.

A dinner of army fare was served in the general mess hall, everyone enjoying it very much. The meeting was a success in

every way, and the attendance was the largest in the history of the Association.

S. J. SCHILLING, *Secretary.*

## SOUTHEASTERN IOWA VETERINARY MEDICAL ASSOCIATION

A meeting of the Southeastern Iowa Veterinary Medical Society was held at the Hotel Brazelton, Mount Pleasant, Iowa, on Tuesday evening, November 22, 1938.

After President J. M. Wilson, of Winfield, called the meeting to order, Dr. Thomas J. McCabe, of Mount Pleasant, presented a paper on "Rabies in Squirrels, Foxes and Skunks." A case report, "Rabies in a Cow," was given by Dr. S. C. Lindsay, of Bonaparte. The discussion which followed was led by Drs. J. W. Giffee, of Cedar Rapids; G. A. White, of Riverside; H. C. Dow, of Fort Madison, and R. V. Beard, of Burlington.

Dr. J. H. Krichel, of Keokuk, presented "Anaphylactic Shock from the Use of Autogenous Mastitis Bacterin in Cattle." "Anaphylactic Shock Following Administration of Blackleg Aggressin" was offered by Dr. H. E. Tyner, of New London. Drs. C. E. Hunt, of Mount Pleasant; E. A. Horner, of Brighton; C. M. Collins, of Ottumwa, and J. D. Reardon, of Galesburg, Ill., participated in the discussion.

Dr. H. M. Griffin, of Morning Sun, presented "Equine Encephalomyelitis Sequelae." Discussion of Dr. Griffin's paper was conducted by Drs. John Patterson, of Des Moines; C. E. Bassler, of Ainsworth, and F. H. Reid, of Washington.

Twenty-three veterinarians from eleven Iowa counties were in attendance at the meeting.

T. J. MCCABE, *Secretary.*

## MISSISSIPPI VALLEY VETERINARY MEDICAL ASSOCIATION

On December 16, 1938, this old Illinois local society held its 34th annual meeting in Galesburg, center of its activities since its organization in 1904. Along with the Keystone (Philadelphia), New York City and Chicago associations, the Mississippi Valley Veterinary Medical Association ranks among the pioneers of local societies.

The program filled out a busy day and evening of papers, talks and illustrated lectures covering topics of current interest. The work of the day was rounded out by a discussion on veterinary education by Drs. Ward Giltner, of Michigan State College, D. M. Campbell, editor of *Veterinary Medicine*, and L. A. Merillat, executive secretary of the American Veterinary Medical Association.

The roll of stated contributions and reporters was as follows:

"Poultry Diseases," Dr. C. A. Brandley, Department of Animal Pathology and Hygiene, University of Illinois.

"Nutritional Diseases," Dr. W. B. Massie, Boston, Ind.

"Rabies and Clinical Problems," Dr. Robert Graham, Department of Animal Pathology and Hygiene, University of Illinois.

"Diseases of Animals and Their Relation to Public Health," Dr. Ward Giltner, Michigan State College.

"Equine Encephalomyelitis," Dr. T. E. Munce, Sioux City, Iowa.

The report on the important results of the surgical operations performed at the Galesburg clinic, in 1938, and the usual question-box plan of bringing out queries of personal significance were distinctive features of the program.

Officers for the ensuing year were elected as follows: President, Dr. Earl R. Kennedy, of Moline; vice-president, Dr. A. R. Cowser, of Farmington; secretary-treasurer, Dr. L. A. Gray (reëlected), of Bushnell; member of Executive Committee, Dr. C. M. Rodgers, of Avon.

Plans for the now famous "Galesburg clinic," in June, and for increasing the program of the annual meeting from one day to two days were approved.

## Bird Refuge for Missouri

Dr. John D. Brock, a physician of Kansas City, Mo., has offered the State of Missouri the use of his 14,000 acres in Oregon County for game propagation. The property, located in the "Big Springs" country, is said to be the largest fenced area in the state. Already it is well stocked with wild turkeys and quail.

# A. V. M. A. HOUSE OF REPRESENTATIVES

Fifth Annual Meeting, New York, N. Y., July 5-8, 1938

| | *Year** | *Delegate†* | *Elected or appointed* | *Alternate* |
|---|---|---|---|---|
| Alabama | Even | I. S. McAdory | 2-26-38 | |
| Arizona | Odd | (R. J. Hight) | 7-2-37 | |
| Arkansas | Even | C. D. Stubbs | 3-7-38 | |
| California | Odd | John L. Tyler | 6-23-37 | |
| Colorado | Even | (A. N. Carroll) | 1-20-38 | |
| Connecticut | Odd | (G. E. Corwin) | 2-3-37 | I. R. Vail |
| Delaware | Even | (R. M. Sarde) | 12-22-36 | C. C. Palmer |
| District of Columbia | Odd | H. W. Schoening | 6-9-37 | |
| Florida | Even | D. A. Sanders | 6-5-38 | |
| Georgia | Odd | (L. J. Kepp) | 7-9-37 | |
| Idaho | Even | (T. A. Elliot) | 6-18-38 | |
| Illinois | Odd | L. N. Morin | 6-3-37 | |
| Indiana | Even | J. L. Axby | 1-27-38 | |
| Iowa | Odd | (C. J. Scott) | 1-22-35 | |
| Kansas | Even | Edwin J. Frick | 1-20-38 | M. P. Schlaegel |
| Kentucky | Odd | W. W. Dimock | 1-26-38 | |
| Louisiana | Even | (E. P. Flower) | 2-26-36 | J. D. Jones |
| Maine | Odd | S. W. Stiles | 6-11-38 | |
| Maryland | Even | L. J. Poelma | 6-30-38 | |
| Massachusetts | Odd | (W. H. Dodge) | 3-24-37 | H. W. Jakeman |
| Michigan | Even | B. J. Killham | 6-29-38 | |
| Minnesota | Odd | (C. F. Schlotthauer) | 1-21-37 | R. L. West |
| Mississippi | Even | R. H. Stewart | 1-24-38 | |
| Missouri | Odd | A. T. Kinsley | 1-25-37 | |
| Montana | Even | W. J. Butler | 5-31-38 | |
| Nebraska | Odd | O. H. Person | 12-9-36 | |
| Nevada | Even | (Wm. R. Smith) | 1-28-38 | |
| New Hampshire | Odd | C. L. Martin | 2-15-38 | |
| New Jersey | Even | R. A. Hendershott | 1-14-38 | |
| New Mexico | Odd | (F. L. Schneider) | 1-8-37 | |
| New York | Even | (R. R. Birch) | 7-17-36 | L. E. Moore |
| North Carolina | Odd | A. A. Husman | 1-29-37 | |
| North Dakota | Even | (T. O. Brandenburg) | 6-27-38 | |
| Ohio | Odd | (E. J. Starbuck) | 1-20-38 | W. R. Krill |
| Oklahoma | Even | (Frank R. Knotts) | 1-11-38 | |
| Oregon | Odd | O. H. Muth | 6-27-38 | |
| Pennsylvania | Even | G. A. Dick | 10-23-36 | Wm. H. Ivens |
| Rhode Island | Odd | (T. E. Robinson) | 1-11-38 | J. S. Barber |
| South Carolina | Even | W. A. Barnette | 1-18-38 | |
| South Dakota | Odd | (G. E. Melody) | 12-11-37 | |
| Tennessee | Even | John H. Gillmann | 1-10-38 | |
| Texas | Odd | M. E. Gleason | 1-21-37 | |
| Utah | Even | Hugh Hurst | 6-24-38 | |
| Vermont | Odd | ‡L. H. Adams | 12-15-36 | |
| Virginia | Even | (P. M. Graves) | 7-1-38 | H. T. Farmer |
| Washington | Odd | E. E. Wegner | 5-23-38 | |
| West Virginia | Even | S. E. Hershey | 10-28-36 | |
| Wisconsin | Odd | Walter Wisnicky | 7-1-38 | |
| Wyoming | Even | (H. D. Port) | 6-28-38 | |
| Veterinary Corps | Odd | R. J. Foster | 12-31-37 | |

*Each organization or geographical unit having representation in the House chooses its delegate and alternate each even-numbered or odd-numbered year, as provided in section 15 of article V of the Constitution.

†Delegates whose names are shown in parentheses were not in attendance at the meeting in New York. Alternates whose names are shown in the column headed "Alternates" were in attendance at one session or both.

‡Deceased.

# NECROLOGY

## GEORGE A. POSSE

Dr. George A. Posse, of Tifton, Ga., died on September 20, 1938. At the time of his death, he was stationed at Tifton on meat inspection service for the U. S. Bureau of Animal Industry.

Born at Del Norte, Colo., February 18, 1900, Dr. Posse attended local grade and high schools, and then entered Colorado State College for the study of veterinary medicine. He was graduated in 1924, and immediately entered the service of the U. S. Bureau of Animal Industry. He was assigned to meat inspection in Chicago and, subsequently, was assigned to the same work in Topeka, Kans., Lincoln, Neb., and Mason City, Iowa, before being sent to Georgia.

Dr. Posse joined the A. V. M. A. in 1924.

## KENNETH JOSEPH MOYE

Dr. Kenneth J. Moye, of Cabool, Mo., died suddenly of a heart attack, on October 16, 1938, while making a professional call on a farm near Cabool.

Born at Las Vegas, N. Mex., on July 27, 1891, Dr. Moye attended local grade and high schools and Colorado College before entering the Kansas City Veterinary College. He was graduated in 1914 and entered practice at Hays, Kan. He had 21 months of military service during the World War, part of the time being overseas. He reëntered practice at Keytesville, Mo., later going to Sayre, Okla. He then spent two years in the service of the U. S. B. A. I. In July, 1937, he located at Cabool.

Dr. Moye joined the A. V. M. A. in 1921. He is survived by his widow (née Grace M. Dyer) and one brother.

## STUART N. CHAPIN

Dr. Stuart N. Chapin, of Battle Creek, Mich., died at his home on October 21, 1938, after a long illness.

Born at Hickory Corners, Mich., on January 16, 1894, Dr. Chapin was graduated from the McKillip Veterinary College in 1919. During the World War, Dr. Chapin was stationed at the Auxiliary Remount Depot 320, at Camp Custer. Following his discharge he entered the employ of the Sullivan Milk Producers Company and worked for the concern for 18 years.

Dr. Chapin is survived by his widow, one son, one daughter, his parents, four sisters and six brothers.

## JAMES D. KOPP

Dr. James D. Kopp, of Davenport, Neb., died on April 1, 1938, of heart disease. He was a graduate of the Kansas City Veterinary College, class of 1913, and practiced for a number of years at Davenport. For the past 15 years, he had been in the service of the Nebraska Bureau of Animal Industry, engaged in field work. He is survived by his widow and two daughters.

## CHARLES H. GODDARD

Dr. Charles H. Goddard, of Pine Village, Ind., died at his home on November 15, 1938, following a heart attack.

Born at Rushville, Ind., in 1875, Dr. Goddard attended grade and high schools. When well past 40, he decided to study veterinary medicine and entered the Indiana Veterinary College. He was graduated in 1921 and located at Pine Village.

Dr. Goddard joined the A. V. M. A. in 1924. He is survived by his widow (née Margaret Wagner), two sisters and three brothers.

## WALTER E. NEARY

Dr. Walter E. Neary, of Watertown, S. Dak., died at his home on November 6, 1938, as the result of an accidental gunshot wound.

Born at Cumberland, Iowa, August 19, 1894, Dr. Neary attended local grade and high schools and then took a course in Saint Benedict's Preparatory School, at Atchison, Kan., before entering Colorado State College for the study of veterinary medicine. He was graduated in 1921 and entered the service of the U. S. Bureau of Animal Industry the same year. At various times, he was stationed at Boise and Moscow, Idaho, and he was inspector-in-charge at the plant of Swift & Company, in Watertown.

Dr. Neary joined the A. V. M. A. in 1923. He was a member of the National Association of B. A. I. Veterinarians and the Twelfth International Veterinary Congress. He was a Mason, a member of the American Legion, and held membership in a number of civic organizations. Surviving are his widow (née Ruth Clay), one son, his mother, two brothers and two sisters. Interment was at Grant, Iowa.

C. H. F.

## WILLIAM H. MURPHY

Dr. William H. Murphy, of Brookline, Mass., died on August 7, 1938, of a heart attack. He was in his 62nd year. He was a graduate of the Ontario Veterinary College, class of 1899, and was City Veterinarian of Boston at one time.

## NATHANIEL T. CLARK

Dr. Nathaniel T. Clark, of Valparaiso, Ind., died at his home on November 16, 1938, after an illness of nine months.

Born in Mount Bridges, Ont., Can., on June 23, 1868, Dr. Clark was graduated from the Ontario Veterinary College in 1892. He practiced for a year in Buffalo, N. Y., then went to Valparaiso and remained there until his death, except for the year 1900-01, which he spent at the McKillip Veterinary College, in Chicago.

Dr. Clark served as a trustee of Center Township from 1931 to 1935. He is survived by his widow, three sisters and a brother.

## WILLIAM J. BRANDEWIE

Dr. William J. Brandewie, of Kalispell, Mont., died November 30, 1938, of a heart attack.

Following his graduation from the Ohio State University, in 1909, Dr. Brandewie served for one year, as a graduate assistant in the Department of Surgery, of the College of Veterinary Medicine. After he completed this assistantship, he entered private practice in Columbus, Ohio. Several years later, he went to Kalispell, Mont., where he owned and operated a silver fox farm. He is survived by his widow (née Edith Knoepke) and two sons.

## JOHN S. ANDRADE

Dr. John S. Andrade, of Huntsville, Ala., died suddenly at his home on December 7, 1938, of heart disease. He was a graduate of the McKillip Veterinary College, class of 1910.

Dr. Andrade joined the A. V. M. A. in 1911. He served as A. V. M. A. resident secretary for Alabama (1922-23). He was president of the Alabama State Veterinary Medical Examining Board for many years and was prominent in veterinary medical circles throughout the South.

R. L. M.

## WILLIAM H. STEPHENSON

Dr. William H. Stephenson, of Stockton, Ill., died shortly after being injured in an automobile accident on December 7, 1938, near Warren, Ill.

Born on the family homestead near Apple River, Ill., on October 30, 1875, Dr. Stephenson was graduated from the Chicago Veterinary College in 1905. He practiced first at Warren, Ill., with the late Dr. E. K. Kane (Ont. '91). In 1908, he moved to Stockton, having purchased a practice there. Since 1912, Dr. Stephenson had

practiced in partnership with Dr. James B. Baber, his brother-in-law. The survivors include the widow (née Lulu N. Gann), one sister and five brothers.

## FRANCIS H. McCLEAN

Dr. Francis H. McClean, of Neoga, Ill., died on December 8, 1938, while making a professional call on a farm three miles south of Neoga. A heart attack was the cause of death.

Born at Neoga, June 11, 1888, Dr. McClean attended local grade and high schools and then entered the Terre Haute Veterinary College. He was graduated in 1913 and returned to Neoga, where he engaged in general practice with his father, Dr. J. W. McClean. During the World War, the younger McClean was commissioned as a second lieutenant in the Veterinary Corps, and was stationed at Camp Greenleaf and Fort Sill.

Dr. McClean joined the A. V. M. A. in 1918. He is survived by his widow, two daughters and his father.

## HERMAN A. VETTERLING

Dr. H. A. Vetterling passed away at his home in Huron, S. Dak., on December 11, 1938, after an illness of several months.

Born in McPherson, Kan., on April 24, 1883, Dr. Vetterling moved, when a small boy, with his parents to a ranch near Wetonka, S. Dak. He attended local grade and high schools, and then entered the University of Minnesota, where he spent one year. Later, he attended the University of San Francisco, and then decided to study veterinary medicine. He received his veterinary degree from the State College of Washington in 1920. He entered practice at Wetonka, S. Dak. and, in the summer of 1928, moved to Wessington, S. Dak., where he practiced until 1934. Most of the time from that date until April, 1938, he spent in the employ of the U. S. Bureau of Animal Industry on tuberculosis eradication work. When he became ill in April and was unable to continue in this work, he moved to Huron.

Dr. Vetterling joined the A.V.M.A. in 1935. He was a member of the South Dakota Veterinary Medical Association. He is survived by his widow, one son, his mother and three sisters. G. E. M.

## STEPHEN H. SWAIN

Dr. Stephen H. Swain, leader among the early non-graduate veterinarians of central Illinois for more than 50 years, died at his home in Decatur, December 19, 1938, at the age of 97. The deceased was a prominent figure in the work of the Illinois Veterinary Medical and Surgical Association, a society of non-graduates which flourished in that state during the nineteenth century, while the association of graduates, the Illinois State Veterinary Medical Association, was still a fledgling struggling for membership, because graduates were few and the spirit of organized veterinary medicine was yet to develop.

It is to the credit of Doctor Swain and his peers that they organized for the temporal betterment of their lot and that they kept abreast of the times by reading contemporary literature, perhaps more incessantly than many of their immediate successors who overlook these essential traits.

The older members of the present Illinois association will recall that Doctor Swain and his organization aided not only in having the veterinary practice act of that state passed by the legislature, but also in guarding against the licensing of incompetent intruders thereafter.

## JAMES HARRISON

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Born in Toronto, Canada, on March 22, 1864, Dr. Harrison was graduated from the Ontario Veterinary College in 1890. He practiced in Kalamazoo, Schoolcraft and Galien, Mich., before going to Buchanan in 1935.

Dr. Harrison joined the A. V. M. A. in 1910. He is survived by his widow (née Ruth E. Parker) and two children by a former marriage.

## JOSEPH W. IRELAND

Dr. Joseph W. Ireland, of Quincy, Ill., one of the pioneer members of the Illinois State Veterinary Medical Association, passed away at Blessing Hospital in that city, on December 17, 1938.

Born at King, Ontario, August 15, 1861, Dr. Ireland was graduated from the Ontario Veterinary College in 1885, and located in Belvidere, Ill., the following year. In 1899 he moved to Quincy and, with the exception of six years (1926-1932) while employed in the research department of the Moorman Manufacturing Company, he was engaged in practice.

Dr. Ireland was a member of the A. V. M. A. from 1926 to 1936. He is survived by his widow (née Ida Ann Root) one daughter, one son and one sister.

## CHARLES HENNING JOHNSON

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## Dangerous Drugs Under Scrutiny

Under date of September 8, 1938, Mr. W. G. Campbell, Chief of the U. S. Food and Drug Administration, made public the opinion of the federal authorities relative to several drugs, the promiscuous sale and use of which are believed to be attended by certain risk.

Although aminopyrine has been employed as a drug for more than 40 years and although agranulocytosis has been recognized as a clinical entity for the past 16 years, the rôle of aminopyrine as probably the most important causative factor in agranulocytosis was not recognized until about six years ago. Once the causal relationship between the drug and the disease was suspected, confirmatory evidence rapidly accumulated and was reported in medical literature. There is now no doubt that this drug has been responsible for numerous deaths in the United States.

Since the introduction of cinchophen as a therapeutic agent some 30 years ago, many reports of its toxic manifestations have been reported in medical literature. These include numerous cases of acute yellow atrophy and cirrhosis of the liver which result in permanent damage and not infrequently in death. The dangerous potentialities of this drug are now generally recognized by informed physicians. The toxic properties of neocinchophen are generally similar to those of cinchophen.

In the light of these facts, careful consideration has been given to the status of aminopyrine, cinchophen and neocinchophen, under the currently effective provisions of the Food, Drug and Cosmetic Act which deal with traffic in dangerous drugs.

In the opinion of the Food and Drug Administration, aminopyrine, cinchophen and neocinchophen, and drug preparations containing them, when found in interstate commerce under labeling which may result in their use by the general public, are actionable under Section 502(j) of the new Food, Drug and Cosmetic Act.

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practiced in partnership with Dr. James B. Baber, his brother-in-law. The survivors include the widow (née Lulu N. Gann), one sister and five brothers.

## FRANCIS H. McCLEAN

Dr. Francis H. McClean, of Neoga, Ill., died on December 8, 1938, while making a professional call on a farm three miles south of Neoga. A heart attack was the cause of death.

Born at Neoga, June 11, 1888, Dr. McClean attended local grade and high schools and then entered the Terre Haute Veterinary College. He was graduated in 1913 and returned to Neoga, where he engaged in general practice with his father, Dr. J. W. McClean. During the World War, the younger McClean was commissioned as a second lieutenant in the Veterinary Corps, and was stationed at Camp Greenleaf and Fort Sill.

Dr. McClean joined the A. V. M. A. in 1918. He is survived by his widow, two daughters and his father.

## HERMAN A. VETTERLING

Dr. H. A. Vetterling passed away at his home in Huron, S. Dak., on December 11, 1938, after an illness of several months.

Born in McPherson, Kan., on April 24, 1883, Dr. Vetterling moved, when a small boy, with his parents to a ranch near Wetonka, S. Dak. He attended local grade and high schools, and then entered the University of Minnesota, where he spent one year. Later, he attended the University of San Francisco, and then decided to study veterinary medicine. He received his veterinary degree from the State College of Washington in 1920. He entered practice at Wetonka, S. Dak. and, in the summer of 1928, moved to Wessington, S. Dak., where he practiced until 1934. Most of the time from that date until April, 1938, he spent in the employ of the U. S. Bureau of Animal Industry on tuberculosis eradication work. When he became ill in April and was unable to continue in this work, he moved to Huron.

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# GLEANINGS FROM OUR CORRESPONDENCE

## BIRTHS

To Dr. and Mrs. F. F. McNeely, of Colfax, La., a daughter, Mary Lavinia, October 12, 1938.

To Dr. and Mrs. Ralph R. Younce, of Tremonton, Utah, a daughter, Patty Rae, November 16, 1938.

## MARRIAGES

Dr. Maurice S. Shahan (Colo. '24), of Washington, D. C., to Miss Gladys S. Mattison, of Rice Lake, Wis., December 3, 1938.

Dr. James A. Frazee (U. P. '37), of Clinton, N. J., to Miss Harriot Van Langeveld, at Philadelphia, Pa., October 29, 1938.

## PERSONALS

Dr. H. M. Mershon (U. P. '38) has entered private practice at Linesville, Pa.

Dr. Frank E. Allen (O. S. U. '36), formerly of Pasadena, Calif., is now located in Van Nuys, Calif.

Dr. Wilson M. Pang (Wash. '37), formerly of Honolulu, reports a new address: Wailuku, Maui, T. H.

Dr. Willem Dekker-van Ghyl (K. S. C. '35) reports a change of address from Laurel, Md., to Riva, Md.

Dr. W. Walter Martin (U. P. '95), of Spring Lake, N. J., is spending the winter in Miami, Fla., as usual.

Dr. J. H. Williams (Colo. '31) has changed locations from Imperial, Calif., to Fall River Mills, same state.

Dr. E. G. Pringle (Mich. '32), of Goshen, Ind., is planning to build a new hospital just west of his new home, a quarter-mile west of Goshen on U. S. 33.

Dr. Carl R. Benton (Mich. '36), of Salem, Mass., has moved his hospital and clinic to 392 Lafayette St., same city.

Dr. James A. Frazee (U. P. '37) announces a change of address from Annandale, N. J., to 114 Center St., Clinton, N. J.

Dr. John A. Ziebarth, Jr. (Wash. '23), city veterinarian of Pocatello, Idaho, recently completed a modern, small-animal hospital.

Dr. Ralph R. Younce (Colo. '35) has given up his practice at Tremonton, Utah, to accept an appointment with the CCC at Vancouver, Wash.

Dr. A. W. Groth (St. Jos. '20) has resigned from the service of the U. S. Bureau of Animal Industry. He is planning to enter practice at a future date.

Dr. C. C. Stewart (Colo. '13), of Colorado Springs, Colo., actively campaigned against the "Basic Science Bill," which was debated in the recent elections in Colorado.

Dr. J. E. Wittrock (Wash. '37), a member of the B. A. I. force in Idaho Falls, Idaho, was awarded a medal for winning the recent amateur golf championship of southeastern Idaho.

Dr. Chas. H. Ozanian (O. S. U. '38) is employed by the Reliance Dairy Farms, at Downey, Calif. The dairy has a herd of 2,000 cows.

Dr. David Splaver (O. S. U. '37) has moved from Tracy, Calif., to 437 S. Church St., Visalia, Calif., where he is engaged as a live stock inspector for the California State Department of Agriculture.

Dr. W. L. Pinckard (A. P. I. '37), who was formerly associated with the Knoxville Veterinary Hospital, Knoxville, Tenn., has moved to Elizabethton, Tenn., where he has entered private practice.

Dr. Ronald Gwatkin (Ont. '19), who has been with the Ontario Research Foundation in Toronto, Ontario, for several years, is now located at the Veterinary Research Station, Lethbridge, Alberta.

Dr. H. P. Galloway (Colo. '38), of Cortez, Colo., was in charge of the hospital and practice of Dr. T. I. Means, of Santa Fe, N. Mex., while the latter was attending the Army Veterinary School in Washington, D. C.

Dr. H. E. McMillan (Ont. '08), of Rupert, Idaho, who suffered a broken ankle while taking a blood-sample in connection with the Bang's disease program on October 15, is reported to be recovering satisfactorily.

Dr. Glenn Holm (Iowa '36), who was engaged in general practice at Rexburg, Idaho, has moved to Moscow, Idaho, having accepted an appointment in the Department of Bacteriology of the University of Idaho, Moscow.

Dr. Edward C. Preston (U. P. '37), formerly of Philadelphia, Pa., is now located at 11 Division St., Newton, N. J., where he is in charge of the Sussex County Artificial Breeding Unit of the New Jersey Holstein Friesian Association.

Dr. John D. Vietti (Wash. '38) has resigned his position as junior veterinarian with the B. A. I. force in Puyallup, Wash., and has moved to Los Banos, Calif., where he is employed by the State of California as a live stock inspector.

Dr. Clare W. Pritchard (Wash. '32), of Pasadena, Calif., is now practicing in partnership with his brother, Dr. Robert M. Pritchard (Wash. '30). They are building a small-animal unit in Palm Springs as an auxiliary to their hospital in Pasadena.

Dr. Jay H. Bouton (Colo. '25), of Aurora, Colo., gave a number of radio talks over Station KFEL, opposing the passage of the so-called "Health Freedom Bill," which was before the Colorado voters on November 8. It was defeated by a large majority.

Merry Christmas

and a

Happy and Prosperous

New Year

ASHE LOCKHART, INC.